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A SERIES OF CASES RESEMBLING LARYNGEAL DIPHTHERIA.[1]

By FRANK BEARE, M.D.,
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DURING the winter of 1929 in Adelaide there occurred a fairly large epidemic of infections presenting certain features that would seem to be unusual and worthy of record. The observations here recorded have been made both in my own practice and at the Infectious Diseases Department of the Adelaide Hospital. A total of 52 patients has been observed, 15 in my private practice and 37 in the Adelaide Hospital. It would seem that the condition is either an entirely new clinical entity or that it is a condition existing before, but now occurring in a much more severe form. In this respect several general practitioners of very large experience have stated that they have never previously encountered anything quite like it.

Briefly the condition is as follows. It commences as an upper respiratory infection characterized by running from the nose, sneezing, sore throat and slight cough with only a moderate rise in temperature and pulse rate and very little general distress. The illness gradually passes into a more serious complaint. The cough becomes more troublesome and definitely "croupy" or "brassy" in character, practically no sputum is expectorated, even in persons old enough to do so. In many cases the voice becomes hoarse and occasionally there is definite aphonia. The respiration is of a definitely stridulous nature, in some cases being heard some considerable distance away from the patient. Fairly rapidly the breathing becomes more and more difficult and distressed with varying degrees of indrawing of the abdomen, lower thoracic walls and intercostal spaces with inspiration. All the extraordinary muscles of respiration are seen to be strongly in action. The patient, especially when young, becomes very distressed and very often is obviously terrified, becoming extremely restless and difficult to manage. In many cases, cyanosis varying from a heliotrope tint to a deep blue colour occurs. With the onset of the graver obstruction to respiration the temperature rises to 37·8° C. (100° F.) or so, the pulse becomes more rapid and the respiratory rate increases. There is now obviously more malaise and all inclination to eat goes. When food, either solid or fluid, is taken under persuasion, there is obvious pain on swallowing. On physical examination practically no other abnormality, except a few rhonchi and the noise of the difficult breathing heard in the lungs, is found. The heart, except for its rapid action, is clinically normal. The pharynx is almost invariably intensely reddened and swollen in appearance. The tonsils are as a rule enlarged and very red; on some occasions they have been covered with a thin confluent yellowish exudate. Occasionally the cervical lymphatic glands have been felt enlarged and tender. The nose in nearly all cases has contained non-sanguineous muco-pus. No excoriation of the upper lip or any *herpes febrilis* has been seen. Atmospheric conditions do not seem to influence in any way the course of the disease, nor did the condition seem to be worse during the night than the day time.

The course of the illness varies. Possibly after several days of distress, as outlined above, the condition gradually becomes less and less severe, the respiratory obstruction becomes less apparent, the stridor not so severe, the cough less "croupy" and the general condition of the patient more comfortable. After three or four days or so all that remains is a harsh cough becoming "croupy" on exertion or in the presence of some emotional stimulus. Finally the cough dies down and the individual becomes quite well again.

On the other hand, in the minority of instances the cough becomes more distressing, the respiratory difficulty more pronounced, the cyanosis deeper, the restlessness more intense, the pulse more rapid and weaker, profuse sweating breaks out and death ensues more from a circulatory failure than a respiratory breakdown. As a rule for some time before death there appears to be an abatement of the respiratory distress but an increasing circulatory embarrassment. Finally the picture may be that of a fairly quiet respiration, an alert mind and a sudden death on attempting to sit up or after a moderate coughing seizure.

The following notes will illustrate the above forms of the illness.

CASE I. F.W., aged six years, was taken ill on May 12, 1929, with "cold and sore throat, running from the nose and a croupy cough." The breathing was noticed to be distressed by the parents. On May 14, 1929, the temperature was 36·9° C. (98·4° F.), the pulse rate 116 and respiratory rate 26 to 36 per minute. The boy had a croupy cough with stridulous breathing. He was restless and resented being examined. There was moderate indrawing of the abdomen and lower thoracic walls with inspiration. The throat was reddened and the tonsils were slightly enlarged, but no membrane was present. No other abnormality was detected. He was placed in a steam tent, given warm drinks and an expectorant mixture. Thirty thousand units of diphtheria antitoxin were administered intramuscularly. The next day he was comfortable, except for a slight croupy cough and very slight retraction of the abdomen on breathing. Two days after this he was well except for a croupy cough and three days later again was quite well. All examinations for Klebs-Löffler bacilli were without result.

CASE II illustrates the fatal type. L.P., aged three years and three months, had a croupy cough and a running nose on July 13, 1929. On July 15, 1929, in the evening, difficulty in breathing was noticed. On July 16, 1929, the temperature was 37·6° C. (99·8° F.), the pulse rate 140 and respiratory rate 40 per minute. The child was breathing stridulously, the lips were cyanosed and the patient looked very ill. The tongue was dirty, the throat red and the tonsils were enlarged; no membrane was present. There was severe indrawing of the abdomen, thoracic walls and supraclavicular regions with inspiration. Intubation was attempted, but the tube was not retained, so tracheotomy was performed. The child was placed in a steam tent and oxygen was administered. Thirty thousand units of diphtheria antitoxin and thirty cubic centimetres

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 27, 1930.

of antistreptococcal serum were injected intramuscularly and an expectorant mixture was given by mouth. There was a slight improvement in colour after these procedures, but the child still remained exceedingly toxic. Later the girl became more cyanosed, the breathing became weaker and finally she died early in the morning of July 17, 1929. A laryngeal swab, taken during life, yielded a pure culture of *Staphylococcus aureus* and from the material obtained by tracheal puncture a *Staphylococcus aureus* and a hæmolytic streptococcus were grown. At autopsy the following lesions were found. The trachea from the cranial end to the bifurcation showed an intensely congested surface covered with a purulent sanguineous exudate, but no membrane. From this region a *Staphylococcus aureus* and a hæmolytic streptococcus were grown. The glottis was slightly swollen as were the aryteno-epiglottidean folds. From this region a *Staphylococcus aureus*, a *Streptococcus hæmolyticus* and a pneumococcus were grown. The left lung was considerably collapsed, most of the upper lobe being airless and of a dark purple colour. The posterior two-thirds of the uppermost lobe of the right lung were purple and nearly airless, the anterior third being emphysematous. The middle lobe contained some areas of collapse, while the lowermost lobe appeared to be normal. From the lungs a *Staphylococcus aureus*, hæmolytic streptococcus and pneumococcus were grown. Microscopically the compressed parts of the lungs showed congested blood vessels, a moderate number of polymorphonuclear cells in some of the compressed alveoli and a generally airless condition of the tissues. The liver and kidneys showed a certain degree of cloudy swelling, while the suprarenals were congested and had many degenerated cells in the medulla. On culture the blood yielded staphylococci, probably *albus*.

The changes found at autopsy, outlined in the latter case, were typical of the majority of the five cases that came to *post mortem* examination. Briefly these may be summarized as follows: Intense inflammatory changes in the upper air passages, more especially the trachea, evidence of obstructed breathing, such as patchy collapse of the lungs with compensatory emphysema and manifestations of the action of a toxin on the organs.

It was possible to investigate bacteriologically eleven of the cases in the series and the findings may be summarized as follows: In eight cases *Staphylococcus aureus* was found in the larynx, in pure culture on two occasions. The growth on culture medium was always profuse. The three patients not yielding *Staphylococcus aureus* all had pneumococci and one of them a staphylococcus, possibly *Staphylococcus citreus*. In two cases staphylococci were obtained from the blood culture, but they were probably *Staphylococcus albus*, most likely from the skin. From the three patients in whom tracheal puncture was done, a *Staphylococcus aureus* was obtained on two occasions, in one instance in pure culture. At autopsy *Staphylococcus aureus* was found in nearly all parts of the respiratory tract on every occasion. All the patients were examined for the presence of Klebs-Löffler bacillus, but on no occasion was this organism found.

From the ætiological aspect there does not seem to be anything outstanding. In the series infections occurred at all ages from four months to nine years with no special concentration in any one age group. One old lady of eighty-six years of age was included in the series, but no other adult, nor did I observe any example in any way resembling the condition among adults in my practice.

Most of the cases occurred in May and June and ceased altogether in early August.

It seems to me that the condition should be regarded as communicable. Several observations were made in which the condition occurred in two or more members of the same family in succession. The most striking example was the following.

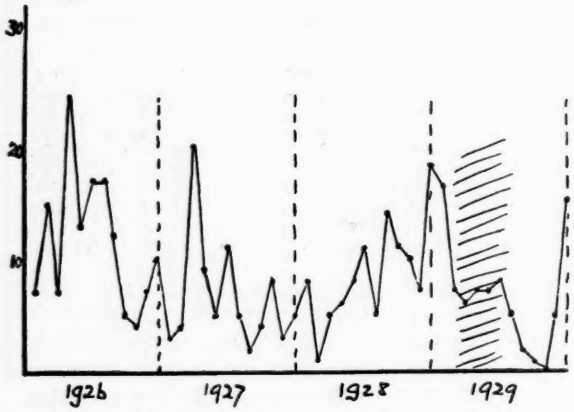
On May 7, 1929, I was called to Thebarton to see a patient, Ray H., suffering from a typical example of the condition. He was sent into hospital. At that time his younger brother, Robert, was quite well, but was sent away to the grandmother's home in Lower North Adelaide. On May 13, 1929, I was called to Lower North Adelaide, where I found this Robert H. suffering from the condition and sent him into hospital. The next day I was called to see Jack H., a cousin at the same home in Lower North Adelaide, and sent him into hospital with the condition.

From the latter observations the incubation period would seem to be a few days, the time usually given as the incubation period for a staphylococcal infection.

From the above it is obvious that the condition is very similar to laryngeal diphtheria of the primary type, that is, laryngeal diphtheria, commencing in the larynx and not with any obvious naso-faucial involvement. That this group of infections was not diphtheria is borne out by the fact that not one nose nor one throat swab, positive for the Klebs-Löffler bacillus, was obtained from these patients. Moreover, swabs taken from the larynx, from the tracheotomy wound of those patients in whom this operation was performed, or from the material sucked up in a needle introduced into the trachea by means of a tracheal puncture, in every instance failed to yield the diphtheria bacillus. In addition, swabs taken from various parts of the respiratory tract at autopsy were always similar in this respect.

From what I can gather from other medical men with whom I have discussed the facts, and from the literature, it seems that it is unusual for ordinary cases of "croup" or *laryngitis stridulosa* to be so severe or to end fatally. The mortality rate in this series of 51 infections was 17% of the hospital patients. Further, *laryngitis stridulosa* does not occur in epidemic form—there must have been hundreds of cases of the condition I have described in Adelaide last winter. Again, energetic treatment by means of diphtheria antitoxin in large doses—a minimum of twenty thousand and a maximum of one hundred thousand units—given both intramuscularly and intravenously, was of no apparent benefit. The mortality rate, 17%, in this series was very much higher than is found with diphtheria. During the last seven years 819 patients with diphtheria have been treated at the Adelaide Hospital with 38 deaths, that is a mortality rate of 4·6%. Moreover, supposing, improbable as it may seem, that these infections did happen to be all examples of primary laryngeal diphtheria which gave normal swabs, one would expect a sudden rise in the incidence of faucial diphtheria during the period in which this series occurred. That this

was not so is shown by the accompanying graph which shows the number of admissions for diphtheria to the Adelaide Hospital month by month over the last four years.



Graph showing the admissions to the Adelaide Hospital of patients suffering from diphtheria, month by month, during the period 1926 to 1929.

Lastly, in no case in which a tracheotomy was performed or an autopsy carried out, was the slightest suspicion of a membrane found. Therefore, I think it is safe to assume that this series did not consist of cases of laryngeal diphtheria, but consisted of cases of a condition closely resembling that condition in its clinical features.

I have searched the literature for a description of a group of cases such as the above, but have found very little. In 1919 P. S. Messent[1] records that during an epidemic of pneumonic influenza at Broken Hill, New South Wales, there were fourteen patients sent into hospital as suffering from diphtheria, either laryngeal or faucial, who proved not to be suffering in this way. Characterized clinically by a croupy cough, difficulty in breathing, stridulous respiration and marked retraction and small patches of membrane on the fauces in some instances, they all failed to yield the diphtheria bacillus on investigation. At autopsy in one case, that of a boy, aged about three years, there was no membrane, but a plum coloured injection of the mucous membranes of the larynx and trachea with œdema of the larynx, more especially of the false cords. Dr. Messent found that tracheotomy was of no avail. Several of his infections proved fatal. At the time Messent pointed out that during the 1890 influenza epidemic the death rate in South Australia from diphtheria was unusually high. He suggested that it was quite possible that this might be explained by the inclusion, amongst the diphtheria group, of infections similar to those he described.

In 1929 R. Y. Mathew[2] described a series of four infections at Toowoomba, Queensland, which resembled laryngeal diphtheria clinically. These were characterized by croup with gradually increasing respiratory obstruction. In none of them was the diphtheria organism isolated, nor did the infections respond to treatment with diphtheria antitoxin. His findings suggest that the condition was primarily an infection of the larynx with highly virulent and highly toxigenic staphylococci. One case came to autopsy when he found that the trachea and bronchi contained some sero-purulent material, but the air passages above the trachea were clear and clean. On the surface of both lungs were areas in which the pleura was dark red in colour and mottled. These areas, when sectioned, appeared to be confluent areas of bronchopneumonia producing a pseudolobar effect. A *Staphylococcus aureus* was grown from these patches in the lungs. In other cases *Staphylococcus aureus* was grown from the throat and material drawn up from the tracheotomy tube. These infections occurred in July and August and he noted that three members in one family were affected in succession. Mathew states that there were numerous examples of this condition in the Darling Downs district at the time.

Wallace King,[3] writing from Dungog in 1929, records having seen an example of a "diphtheria-like disease" in July. The child was two and a half years of age and had two attacks of the condition, one in March and the other in July. In both attacks following a cold there were stridor, croupy cough and well marked supraclavicular and infrasternal recession. On the tonsils there were seen patches of greyish-yellow false membrane. Swabbings failed to yield diphtheria bacilli on each occasion.

It seems to me that similar outbreaks must have occurred elsewhere in Australia at the same time. In fact Dr. von der Borch, of Broken Hill, in a private communication stated that there was a similar group of infections at that place during the midwinter of 1929, but that they were regarded as being due to diphtheria bacilli. Nevertheless, there seems to be some strange association between Adelaide and Toowoomba. It will be remembered that very few cases of the typhus-like disease described by Dr. F. S. Hone occurred in regions other than these two towns.

As regards treatment very little can be said. Placing the child in a steam tent undoubtedly relieves the condition a little. Expectorant mixtures seem to be of some value. The injection of large doses of diphtheria antitoxin in my opinion is not efficacious, especially when one remembers the wonderful effects of that measure in cases of true diphtheria. Antistreptococcal serum was not fully tried, but I do not see how it could do any good, specifically at any rate.

Reasoning on the supposition that the condition was a septicæmia, intravenous gentian violet solutions were tried, but I was not satisfied that it made any difference to the patients on whom it was used. Of one thing I am convinced and that is that tracheotomy does not relieve the respiratory distress one iota. This was done on several occasions and no benefit whatever resulted.

Conclusions.

From my observations I consider that this diphtheria-like disease in children is anatomically mainly a tracheitis. The organism probably arrives there from the nose and throat and from the trachea passes to the larger bronchi, but seemingly does not cause pneumonia. Whether the *Staphylococcus aureus*, so commonly found in this series, is the causative organism, I should not like to say. Lately much suspicion has been thrown on the staphylococci as a fertile cause of disease; this condition may be due to these organisms, but the evidence is far from conclusive. It seems certain that the Klebs-Löffler bacillus is not the causative organism. I have little doubt in my own mind but that the condition is communicable; that it is even more destructive to life than diphtheria is borne out by the mortality rates in this series at any rate. I am sorry that I can offer no more effective suggestions as regards treatment and feel that I have made the lives of my fellow general practitioners more troublesome by drawing their attention to another condition from which laryngeal diphtheria has to be diagnosed in the early hours of the morning.

Acknowledgements.

I should specially like to thank my late house physician, Dr. Gillett, without whose help I could not have carried out these investigations. Dr. Gillett practically lived at the Isolation Block during the two months in which most of these infections occurred, and his very complete case records proved of inestimable value in compiling this paper.

I should also like to thank Dr. L. B. Bull and the staff of the laboratory at the Adelaide Hospital for their efforts in investigating the bacteriological aspects of the condition.

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(1) P. S. Messent: "Secondary Infection in Influenza," THE MEDICAL JOURNAL OF AUSTRALIA, October 25, 1919, page 351.
(2) R. Y. Mathew: "The Staphylococcus Aureus as the Probable Cause of a Fatal Disease Simulating Laryngeal Diphtheria," THE MEDICAL JOURNAL OF AUSTRALIA, January 11, 1930, page 34.
(3) Wallace King: "A Diphtheria-like Disease," THE MEDICAL JOURNAL OF AUSTRALIA, January 11, 1930, page 50.

VACCINE THERAPY.[1]

By D. L. BARLOW, M.S., M.D. (Adelaide),
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THE subject of vaccine therapy is, of course, too large to discuss at all completely in a short paper; therefore I intend merely to refer briefly to certain of the more important practical aspects.

Considerable difference of opinion exists as to the value of vaccines in the prevention and treatment of disease. Imperfect methods of application have given rise to many disappointing results and the position has been further complicated by the commercial exploitation of stock vaccines. It is no exaggeration to say that only in a very small proportion of cases has vaccine therapy been carried out in a thoroughly scientific manner. For such reasons many practitioners have underestimated the value of this method of treatment and this is not surprising. There have been others who have used vaccines on every possible occasion and have greatly overestimated their successes. However, the actual degree of success achieved depends, as in other branches of medical work, on the amount of skill and care employed. Here the importance of proper correlation of the clinical and laboratory activities deserves mention.

If we are to obtain the best possible results, the first step in the use of a vaccine must be a reasonably complete investigation of the condition by a clinical bacteriologist. As a rule he should see the patient and arrange for making the necessary cultures and should be responsible for the composition of the vaccine and not leave this to a technician. He should also advise in regard to the exact way it should be employed, if he is not administering it himself. Such collaboration between the practitioner treating the patient and the bacteriologist is undoubtedly of great advantage to the patient. It is not possible to do much good by rule-of-thumb methods, such as sending out a series of fixed doses, for we cannot be sure in advance how the individual will react to the treatment. If this method is employed, it is certain that harmful effects will not infrequently occur from overdosage, unless the scale of dosage adopted is on such a low plane that in most cases failure is assured from underdosage. The best that can be done seems to be to sketch out an average series of doses and suggest modification of this according to indications as the patient progresses. For this purpose the vaccine can be sent out in bulk, preferably in the special rubber-capped bottles or a number of one or two cubic centimetre ampoules can be filled, from each of which the desired dose can be taken and the remainder discarded.

From what has been said it follows that many cases of failure are avoidable and that it is often possible to do good even when previous efforts have been unsuccessful. It is perhaps scarcely necessary to add that failures will sometimes occur in spite of the greatest care, as there is still much to be learned about this admittedly difficult subject.

Mode of Action of Vaccines.

The mode of action of vaccines cannot be said to be established in detail, but in the main they give rise to a stimulation of the defensive mechanisms of the body which results in the production of antibodies of various kinds, thereby helping to overcome the infecting organism.

It is sometimes argued that the absorption of bacteria and toxins from the site of infection should provide the natural stimulus and that we cannot

[1] Read at a meeting of the South Australian Branch of the British Medical Association on March 27, 1930.

expect to induce the body tissues to make a better response by adding an artificial stimulus. But we are familiar with chronic infections which flare up from time to time. It appears to me that if in the chronic, more quiescent phases the body were making its maximum defensive effort, then any exacerbation must result in disaster.

Anybody who has seen a patient suffering from enteric fever brought to convalescence within forty-eight hours by means of a single dose of vaccine given intravenously, must realize that Nature can be helped to do her best by such means. I do not wish to discuss at this stage the relative merits of this form of treatment of typhoid fever, but it will serve as an illustration of the favourable effects which vaccine therapy may produce even in acute illness. Incidentally it has been shown that the action in this instance is non-specific. Obviously vaccines are powerful weapons which require careful handling, especially in acute or serious infections.

As a rule vaccines are given subcutaneously and it is thought that much of the antibody is produced locally, but it can scarcely be doubted that an appreciable proportion is formed elsewhere after absorption of bacterial products from the site of injection.

Specificity of Vaccines.

The question of the specificity of vaccines is not yet entirely settled, but little doubt exists that it is never absolute. In some conditions, for example pneumococcal infections, it is a very important factor, whereas in others there is practical evidence against any such narrow limitation of action.

Prophylactic Use of Vaccines.

The use of vaccines to protect against specific infections has met with considerable success, notably in typhoid and paratyphoid fevers, plague, pneumonia. Further advances in this direction are certain to occur as the result of the research which is constantly going on. Professor Besredka, of the Pasteur Institute in Paris, has been using a method of administering vaccines by mouth for the prevention of the typhoid and bacillary dysentery groups of infections. He makes the sterilized cultures up in the form of pills with a suitable coating and reports results as favourable as those resulting from inoculation. The chief advantages are the ease of administration and the very mild nature of the reaction. Most people will want further confirmation of its efficacy, but the method has considerable experimental evidence to support it.

Vaccines in Treatment.

Treatment of established disease requires separate consideration of acute and chronic conditions.

In many acute infections, such as urethritis, conjunctivitis, *otitis media*, small doses of vaccine given at short intervals are certainly capable of assisting the appropriate local treatment in more rapidly overcoming the condition and also reduce the liability to complications.

Good results have recently been reported in the treatment of acute pneumonia with immunogens, a variety of vaccine which will be referred to again.

Occasionally, according to my experience, septicæmic conditions will yield to properly managed vaccine therapy and therefore this method should be more generally resorted to at the earliest possible moment in addition to other measures, including serum treatment. In regard to the latter perhaps I may be excused a digression in order to emphasize the importance of using large doses of serum at the outset in the attempt to knock out the infection. This is much more likely to prove effective than a repetition of daily doses of smaller dimensions, for in the latter instance the infection has an opportunity of going ahead in the meantime.

Wound infections can be greatly benefited by autogenous vaccines. In the acute stage daily small doses are indicated at first with gradually increasing intervals.

The beneficial effect of vaccine in whooping cough is now well known, but it is not fully recognized that a freshly prepared autogenous culture is more effective than a stock preparation. Such a culture is not unduly difficult to obtain if correct procedure is adopted.

It would not serve any useful purpose to attempt to make a list of the subacute and chronic conditions which can be successfully treated by means of vaccines. In some of these it is comparatively easy to obtain satisfactory results, but in many others it is usually possible to do so only by the careful application of specialized knowledge; it is in this class of condition that so many disappointments have occurred. The reasons for failure have, however, been little understood and not infrequently the method has been condemned.

Furunculosis and acne are conditions for which the use of autogenous vaccines is well established.

Infections of mucous membranes, such as recurring colds, chronic laryngitis, chronic *otitis media*, respond well to vaccine therapy; so also do chronic bronchitis, bronchitic asthma and certain forms of colitis. The percentage of instances in which rheumatoid arthritis and osteoarthritis yield to autogenous vaccines, is improving with the greater knowledge of the cause of these conditions. Many so-called rheumatic conditions improve with similar treatment if a focal infection can be discovered. Sometimes, of course, they will clear up with attention to the focus, but in many instances it is better also to improve the resistance to the infection by means of a vaccine, for this will hasten recovery and by aiding to eradicate the infection more completely will reduce the liability to recurrence. The following example will serve as an illustration.

A young man, suffering from iritis of recent occurrence, gave a history of a previous attack two years earlier accompanied by a severe naso-pharyngitis. This attack lasted for over three months in spite of treatment and ever since the patient had suffered from rheumatic pains at intervals in the feet. These were giving a good deal of

trouble at the time of examination. An almost pure culture of hæmolytic streptococci was obtained from the naso-pharynx and treatment by autogenous vaccine was immediately instituted. Rapid improvement occurred and the eye was well on the way to recovery in a fortnight. The feet rapidly became free from pain.

Preparation of Vaccines.

The first step in the preparation of vaccines is of the utmost importance, namely the obtaining of the cultures from the infected areas. Whenever possible, it is advisable to have the material collected from the patient at the laboratory and cultivated immediately, but if this is not possible, special precautions are necessary in order to insure that the important pathogenic organisms do not die in transit to the laboratory. Sometimes the only method is to cultivate the material on suitable media at the bedside and arrange to keep them at or about incubator temperature and transmit them to the laboratory without delay. After the lapse of only a few minutes it is often impossible to obtain cultures of the more delicate pathogens from a swab. This applies particularly to gonococci, influenza bacilli, pneumococci and streptococci among the commoner organisms.

It is a distinct advantage to have examined a direct smear from the material from which the cultures are being made, in order to have an idea of the most suitable media to be used. This is not always possible, of course. The importance of sound technique in the cultivation of organisms which require special conditions for their growth, is obvious. The preparation of the media is of the greatest importance in securing successful results and it is my practice to check this personally in all the essential stages.

Difficulty is frequently experienced in deciding whether a particular microorganism is really pathogenic in the particular condition, for on mucous surfaces particularly many organisms may live a saprophytic existence.

Experience in the use of vaccines for similar conditions is of great value and it is in the lack of this that the fallacy of leaving the preparation to a technician lies, however skilled the latter may be. A method of undoubted value has been evolved by Solis Cohen,[1] known as the pathogen-selective technique. It consists essentially in growing cultures on or in media containing some of the patient's blood. It has been found that the growth of many of the microorganisms is inhibited, but that those which are pathogenic in the individual concerned, grow as usual. The latter are used in the preparation of vaccines.

You are all aware of the uncertainty which has existed in regard to focal infections in the past, and of the rather numerous disappointments which have resulted from removing apparent sources of infection. When it is considered how easy it is to overlook a second and more important source of infection, there can be little wonder that results have been so uncertain. The more obvious foci, such as those connected with teeth or tonsils, have come in for a good share of attention, but use of the method mentioned above has indicated that these are at fault in a comparative minority of cases only, whereas important pathogenic organisms frequently reside in the naso-pharynx, colon and gall bladder. The nasal accessory sinuses, urinary and genital organs also provide their quota of infections. When such infections are suspected, the bacteriologist should obtain cultures from all likely areas and test their pathogenicity in the way described. By this means surgical measures or vaccine therapy or both may be indicated with reasonable assurance of success.

In the preparation of vaccines for asthma patients, where these are indicated, my method is to obtain numerous separate pure cultures, usually from the sputum and naso-pharyngeal swabs, but also at times from other sources, and after sterilization to test the patient's reaction to these by intradermic inoculation of a small quantity of each in saline solution. Only those causing very definite immediate or delayed reactions are employed. The reactions depend not only on directly toxic effects, but in some cases on sensitization to bacterial protein.

Administration of Vaccines.

The correct administration of vaccines is not quite so simple a matter as some would have us believe. Yet it is surprising how ready some laboratory workers have been to send out vaccines with instructions how to use them, including dosage, in spite of having had no practical experience. In my experience numerous patients who have been treated on these lines without benefit, have subsequently responded very favourably to properly managed vaccine therapy. In all cases the initial dose is necessarily in the nature of a trial, as patients react so differently one from the other. Therefore, a small dose is indicated, followed two or three days later by one much larger if no appreciable reaction occurs. This process is continued until the desired reaction is obtained, after which the dose is increased by from 20% to 50% each time, according to the results produced. In chronic infections the intervals are gradually increased as the dose becomes larger until from seven to ten days elapse between inoculations.

Reactions to Vaccines.

Reactions are divided into local, focal and general. Local reaction is the result of inflammatory phenomena at the site of injection and gives rise to the formation of antibodies. The occurrence of focal reaction means that interaction of antibody with the infecting agent is taking place at the site of infection and, if not too violent, is generally followed by improvement. General reaction may vary from the mildest degree of lassitude to a state of severe malaise with rise of temperature. The latter, of course, would signify overdosage.

As a rule, moderate local and focal reactions are aimed at without undue general reaction.

Slight redness and tenderness without actual pain are the sort of local reaction expected. As examples of focal reaction I may mention increased expectoration in chronic inflammation of the bronchial tubes and increased engorgement in conditions such as chronic pharyngitis. In arthritic conditions increased pain and tenderness in the joints usually occur.

It is not always easy to decide how long to keep on treatment with a vaccine, but as a rule it is best not to risk discontinuing too soon. Sometimes, especially in mixed infections of mucous membranes, when progress has occurred, but does not appear to be continuing, it is advisable to repeat the bacteriological examination. Certain organisms may have disappeared, whereas others may have become more prominent than at the first examination and a vaccine made from the latter is not infrequently successful in bringing about further improvement.

Apart from unsatisfactory methods there are other possible causes of failure. Amongst these an important one appears to be the inability of a few patients to respond on account of their poor general health as the result of organic disease or prolonged debilitating conditions.

Special Types of Vaccine.

A word on special types of vaccines may not be out of place. Sensitized vaccines are produced by treating the organisms with the corresponding antiserum. Their only advantage appears to be that somewhat larger doses can be given in acute infections.

Detoxicated vaccines are made by dissolving bacteria in sodium hydroxide solution and then precipitating by neutralizing with acid. They apparently cause less reaction in proportion to their bulk, but have not been proved to be more effective than the usual type.

Immunogens consist of the washings from bacterial cultures. The organisms are removed by centrifugalization and filtration and the resulting fluid can be concentrated by partial evaporation over strong sulphuric acid. So far they have been used chiefly as stock preparations and are still on trial.

Stock vaccines have their uses, not only in the production of protein shock, but in acute conditions whilst waiting for the autogenous preparation and in other cases as the next best thing when it is impossible to obtain a vaccine prepared from the patient. A common fault with stock vaccines has been that they have been made from old laboratory cultures which have been kept going by subculturing for many generations. Actually either primary cultures or first subcultures should invariably be used.

Vaccines and Common Ailments.

It may be of interest to consider the application of vaccine therapy to certain common ailments.

Staphylococcal infections of the skin, such as pustules and furuncles, are rapidly benefited in almost every case, provided correct methods are used. The mistake is often made of discontinuing before a sufficiently large dose has been reached.

Eczema accompanied by infection of *Staphylococcus aureus* or *Streptococcus pyogenes* is a strong indication for vaccine therapy, as great benefit is assured.

Repeated colds can in the great majority of cases be abolished or greatly reduced in frequency and intensity by autogenous vaccines. In some people the treatment may need to be repeated after a year or two in order to obtain lasting effect, but the results make it worth while. It is, of course, important to avoid overlooking chronic infection of the accessory sinuses with retention of products. Where operation is required for sinus infection vaccines will assist to clear the trouble more rapidly and recurrence of polypi would be less frequent if vaccines were more commonly employed. In dealing with frequent colds it is important to examine swabs from the naso-pharynx as well as the nasal mucosa.

It is well to keep in mind the possibility of infection being superimposed on pollen sensitization.

Chronic *otitis media* of the type called catarrhal can often be improved as a result of treating the accompanying naso-pharyngitis. In chronic suppuration of the ear the use of vaccines in addition to local measures is of advantage.

When chronic bronchitis exists, it is necessary to be sure that it is not being kept up by infection of the nasal accessory sinuses. If it is, then this factor requires attention first. Complete bacteriological examination is indicated and this entails a considerable amount of work, as many types of streptococci may be present as well as pneumococci, *Micrococci catarrhales*, staphylococci and sundry bacilli. Treatment must be thorough, but is usually of great value. In old standing conditions it is worth while repeating the inoculation early in the next winter. Improvement frequently continues for some time after treatment has been finished.

Asthma is, as you know, frequently accompanied by bronchitis and, if the latter is treated on these lines, the asthma often disappears. It is necessary, however, to discover whether sensitization to foreign protein exists as well as bronchial infection, otherwise, in spite of improvement of the bronchitis, the asthma may persist.

On account of its importance and close relationship to the subject of vaccine therapy, I trust that a short discussion on sensitization will be of interest. Unfortunately the English journals often contain articles and discussions by people who without adequate experience have decided against the importance of sensitization in asthma, although they are obliged to admit it in regard to hay fever. I have recently met some of these men and discussed the matter with them and am able to say that none of them has ever gone into the subject thoroughly enough to be likely to accomplish anything. Incidentally I was able to show that the protein preparations most commonly used are practically

useless. Freeman and Coke who have gone into the matter scientifically, have obtained results which entirely agree with those of careful workers everywhere. It should be realized that the method of testing by placing liquid extracts on epidermal scratches frequently results in missing important reactions and that in any case the list of extracts available is defective in regard to some of the most important so far as we are concerned.

In desensitization we do not aim at producing a reaction and if a little local irritation does occur, we regard it as a signal to proceed more gradually with the dosage. Individuals vary greatly, both in regard to the dosage required and the duration of treatment necessary to desensitize, but persistence is usually rewarded in the more difficult cases and failure generally means inadequacy of treatment.

Vaccines have recently been used prior to operation where resections or anastomoses of alimentary tract are contemplated. The vaccines have been prepared from the fæces.

Similar preparation before the removal of teeth from a very septic mouth is a wise precaution and is capable of preventing much suffering. It is surprising that the advantages of preoperative vaccine therapy have not been realized more in connexion with operations about the mouth, such as resection of portions of the tongue.

Chronic ulcerative colitis is a condition which has been successfully treated of late by means of vaccine therapy and specific serum treatment at the Mayo Clinic.

It is now fairly well recognized that tuberculin has a definite value, but it is worth remembering that secondary infection of tuberculous lesions can often be lessened by the use of autogenous vaccines.

An indirect way of utilizing the advantages of vaccine therapy combined with those of blood transfusion is still more or less on trial in England under the name of immuno-transfusion. The donor is given one or more inoculations of culture from the patient and after an interval the transfusion is performed. Although the results have not been spectacular, there is evidence to suggest that lives have been saved by it which might not have been saved by simple transfusion, although the latter in itself often enables the patient to overcome the infection.

Conclusion.

In conclusion I have tried to avoid wearying you by technical or theoretical discussions and have omitted a number of case histories for the same reasons, but have attempted to convince you that vaccine therapy has not yet received the attention which its possibilities warrant, and to suggest how a more satisfactory state of affairs can be brought about.

Reference.

(1) M. Solis Cohen: "Accentuating Pathogenic Organisms in Culture by Utilising the Inhibitory Influence of Whole Blood," *The British Journal of Experimental Pathology*, 1927, Volume VIII, page 261.

A CRITICISM OF RECENT WORK ON THE PRESENCE OF LEAD IN THE EGG OF THE DOMESTIC HEN.

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During the past five years I have been engaged in the determination of small quantities of metals in biological material. The knowledge gained by this work which has been carried out in the Chemical Laboratory of the Department of Public Health, New South Wales, qualifies me to criticize a number of papers which have been published recently in The Medical Journal of Australia on the presence of lead in the egg of the domestic hen.

The work referred to is that of W. B. S. Bishop, M.Sc., and comprises four papers, three of which are by W. B. S. Bishop, namely:

1. "The Occurrence of Lead in the Egg of the Domestic Hen, Part I," The Medical Journal of Australia, April 21, 1928, page 480;

2. "The Occurrence of Lead in the Egg of the Domestic Hen: Part II," The Medical Journal of Australia, January 26, 1929, page 96;

3. "The Occurrence of Lead in the Egg of the Domestic Hen: Part III," The Medical Journal of Australia, June 15, 1929, page 792; and the fourth an additional note by W. B. S. Bishop, M.Sc., and T. Cooksey, Ph.D., F.I.C.;

4. "The Occurrence of Lead in the Egg of the Domestic Hen: An Additional Note," The Medical Journal of Australia, November 9, 1929, page 660.

These papers deal with the amount of lead present in the egg, the isolation and nature of the lead compound present and the effect of lead injection on the development of the chick embryo.

The value of this work depends on the presence in the egg of an appreciable quantity of lead and it is in relation to the quantity of lead found that this criticism is made.

The amount of lead present in the egg is given as follows:

Shell, 0·1 to 1·6 milligrammes per 100 grammes of wet material.

Yolk, 0·2 to 1·0 miligramme per 100 grammes of wet material.

White, 0·12 to 0·48 milligramme per 100 grammes of wet material.

The relative amount of shell, yolk and white in an egg may be taken as 6:17:31. From these figures the minimum amount of lead found in the yolk and white of one egg was 0·07 milligramme or 0·14 milligramme per 100 grammes of egg and the maximum 0·32 milligramme or 0·65 milligramme per 100 grammes of egg.

To one who is familiar with the amount of lead found in normal tissues, these figures appear extraordinarily high, and it was for this reason that the lead content of a number of eggs obtained from various sources was determined by me.

The results are given in Table I.

TABLE I.

1. Series.	2. Whole Egg in Grammes.	3. Yolk *plus* White in Grammes.	4. Shell in Grammes.	5. Lead Added in Milligrammes.	6. Lead Found in Milligrammes.	7. Lead Present in Milligrammes.
A	—	57	—	0·00	0·00	0·00
	—	50	—	0·00	0·01	0·01
	—	—	7	0·00	0·01	0·01
	—	93	—	0·00	0·01	0·01
	—	—	12	0·00	0·01	0·01
	—	96	—	0·04	0·04	0·00
	—	100	—	0·00	0·00	0·00
	—	96	—	0·02	0·02	0·00
	—	94	—	0·00	0·00	0·00 }
	—	98	—	0·02	0·02	0·00 } Centrifuged
	—	—	27	0·02	0·02	0·00 }
B	114	—	—	0·00	0·01	0·01
	55	—	—	0·01	0·02	0·01
	55	—	—	0·01	0·02	0·01
	—	104	—	0·01	0·02	0·01
	—	—	13	0·01	0·02	0·01
C	—	96	—	0·00	0·005	0·005 Centrifuged
D	53	—	—	0·00	0·01	0·01

In the above table the eggs in series A, C and D are from fowls in different localities and those in B are new laid shop eggs. In columns 2, 3 and 4 are given the weights of the material on which the analyses were made; column 5 gives the amount of lead added to the egg substance before analysis; column 6 the lead found on analysis and column 7 the amount of lead originally present.

From a consideration of these figures it will be seen that the amount of lead present does not exceed 0·01 milligramme and is independent of the amount of egg material taken for analysis. From this it would appear that portion of even this small amount of lead is due to contamination. This belief is further strengthened by the fact that when all filter papers were eliminated as in the last three samples of series A and the sample in series C, the small amount of lead previously found was reduced.

It would appear therefore that the amount of lead normally present in 100 grammes of egg substance (yolk *plus* white) is less than 0·01 milligramme.

The lead in the droppings of the fowls from which the eggs in series A were obtained, was 0·64 milligramme per 100 grammes; if 100 grammes represent the droppings of one fowl for two days, then the lead passed by one fowl per day would be 0·32 milligramme and it is shown in Table I that the eggs from fowls passing this amount of lead contained less than 0·01 milligramme of lead.

If the figures given by Bishop for the minimum and maximum amount of lead found per 100 grammes of egg substance (yolk *plus* white), namely, 0·14 and 0·67 milligramme, are correct, then it may be assumed:

(i) That variations in the amount of lead intake are closely followed by variations in the amount of lead present in the egg; (ii) that the lead taken into the body readily passes into the egg.

If the amount of lead in an egg is influenced by the lead intake, then it should be possible to increase the amount present in an egg by feeding the fowl with lead.

In Table II are given the results obtained with eggs from a fowl which had been fed with lead for a period of fourteen weeks. The lead was administered in the form of solid lead acetate in a pellet of pollard.

TABLE II.

Time Lead Fed in Days.	Amount of Lead Fed per Day in Milligrammes.	Yolk and White in Grammes.	Lead Added in Milligrammes.	Lead Found in Milligrammes.	Lead Present in Milligrammes.	Lead per One Yolk and White in Milligrammes.
0	0·0	105	0·00	0·00	0·00	0·00
0	0·0	96	0·04	0·04	0·00	0·00
4	0·5	100	0·00	0·02	0·02	0·01
7	0·5	100	0·00	0·02	0·02	0·01
12	0·5	95	0·00	0·02	0·02	0·01
16	0·5	96	0·00	0·02	0·02	0·01
26	80·0	—	—	—	—	—
42	80·0	53	0·00	0·02	0·02	0·02
49	80·0	96	0·00	0·06	0·06	0·03
51	80·0	96	0·02	0·09	0·07	0·035
75	80·0	240	0·00	0·10	0·10	0·02
98	80·0	100	0·00	0·08	0·08	0·04

From a consideration of the above figures it will be seen that with a daily lead feed of 0·5 milligramme the lead content of the egg rose from 0·00 to 0·01 milligramme and remained constant at that figure for sixteen days. On the twenty-sixth day of feeding the amount of lead fed was increased to 100 milligrammes of lead acetate, equivalent to 80 milligrammes of lead. The lead content of the egg increased to 0·02 milligramme after sixteen days' feeding and to 0·04 milligramme after seventy-two days' feeding. As only a very small amount of lead ingested by a fowl appears to enter the egg, it is of interest to find out whether such lead passes into the circulation or is simply passed through the body without being absorbed. To determine this the fowl was killed on the ninety-eighth day, four hours after it had received a dose of 80 milligrammes of lead and the amount of lead present in the bones and tissues determined. The distribution of lead in the body was as shown in Table III.

TABLE III.

Portion.	Weight in Grammes.	Lead Found in Milligrammes.	Lead per 100 Grammes in Milligrammes.
Muscle	84·0	0·05	0·06
Gizzard	40·0	0·038	0·10
Liver	65·0	0·125	0·19
Skin of gizzard ..	5·5	0·50	9·10
Femur	10·0	6·25	62·5

As a comparison the distribution of lead in a normal fowl was determined and is given in Table IV.

TABLE IV.

Portion.	Weight in Grammes.	Lead Found in Milligrammes.	Lead per 100 Grammes.	Remarks.
Gizzard ..	36·0	0·00	0·00	Fowl 3 years old
Droppings	43·0	0·88	2·05	Fowl 3 years old
Femur ..	3·5	0·49	14·0	Fowl 3 years old
Femur ..	4·7	0·74	15·8	Fowl 4 years old

From the amount of lead found in the bones of the fowl which had been fed on lead and the amount in the bones of a normal fowl, there is little doubt that portion of the lead ingested is absorbed into the body and a certain amount deposited in the bones. These figures go to show that lead even when fed to a fowl in large amounts, that is, in amounts greatly in excess of that which the fowl would ingest under normal conditions, does not readily enter the egg. This is in entire agreement with the results given in Table I which show that the amount of lead in an egg from a fowl living under conditions where the amount of lead ingested is small, is less than 0·01 milligramme. Since the figures given in this paper and those given by Bishop are not in agreement, the error must lie somewhere in the methods of analysis employed.

The method employed for the determination given here is as follows:

Fifty to one hundred grammes of egg material (yolk *plus* white) are placed in a platinum dish and heated over a small rose burner until well charred. The char is then ground in a mortar and again heated until all fuming has ceased; it is then extracted with twenty cubic centimetres of one part of hydrochloric acid to two of water on a water bath for three hours. The acid extract is separated from the char by decantation through nine centimetre filter paper and the char washed twice with distilled water. The char is then returned to the platinum dish and the carbon completely burnt off at as low a temperature as possible. The small amount of ash left is treated with ten cubic centimetres of one part of hydrochloric acid to two of water for two hours and added to the first filtrate. The combined filtrates are then evaporated to dryness, taken up in twenty-five cubic centimetres of water, made slightly acid to methyl orange, 0·1 milligramme of copper added and sulphuretted hydrogen passed until a definite precipitate forms. The contents of the beaker are then transferred to a test tube and centrifuged for fifteen minutes. The clear liquid is siphoned off and the precipitate washed with twenty cubic centimetres of sulphuretted hydrogen water and again centrifuged. After siphoning off the clear liquid the precipitate is dissolved by the addition of 0·5 cubic centimetre of nitric acid and evaporated to dryness. The residue is dissolved in 5 cubic centimetres of water *plus* 0·5 cubic centimetre of one part of hydrochloric acid to ten of water. Caustic soda, one in seven of water, is then added until the solution is just acid to methyl orange and the whole made up to ten cubic centimetres.

The lead present in five cubic centimetres of this solution is then determined by comparing the turbidity produced on the addition of one cubic centimetre of a freshly prepared 10% solution of sodium bisulphite with those produced in a series of standards containing known amounts of lead.

The method employed by Bishop is briefly as follows:

The material is dried as far as possible in a hot air oven and then ashed at a temperature which does not exceed dull redness. The ash is then extracted with hydrochloric acid, the extract evaporated to dryness, the residue taken up in water made just acid to methyl orange and sulphuretted hydrogen passed for fifteen minutes and allowed to stand overnight. The contents of the beaker are then transferred to a test tube and centrifuged. The clear liquid is siphoned off and the precipitate washed with sulphuretted hydrogen water. The precipitate is then dissolved by the addition of one drop of nitric acid, the solution evaporated nearly to dryness, one cubic centimetre of sulphuric acid (concentrated) added, heated to strong fuming, cooled and four cubic centimetres of a mixture of water and alcohol in the proportion of three to one added and allowed to stand overnight. The lead sulphate precipitate is separated by means of a centrifuge washed with mixture of sulphuric acid, alcohol and water and finally dissolved in five cubic centimetres of hot ammonium acetate solution, one in two. To this solution are then added one cubic centimetre of ammonium hydrate, one cubic centimetre of 10% potassium cyanide solution and lastly two drops of sodium sulphide solution. The amount of lead present is then determined by comparing the density of colour produced with the density of colour of a standard to which a known amount of lead has been added.

In the method described by Bishop there are two points which are open to question and which have a tendency to give low results. Firstly, it is improbable that the whole of the lead is extracted from the char by treatment with hydrochloric acid as described and secondly the probability of loss through solubility when so small a quantity of lead is precipitated as lead sulphate.

In regard to the first point, the figures given by Bishop and Cooksey in "An Additional Note on the Occurrence of Lead in the Egg of the Domestic Hen," THE MEDICAL JOURNAL OF AUSTRALIA, November 9, 1929, indicate that all the lead is not extracted by treatment of the char with hydrochloric acid and therefore no further comment is necessary here.

In regard to the second point, a series of determinations was carried out by me which shows that a small amount of lead is lost when the lead is

precipitated as the sulphate. For these experiments six twenty-five cubic centimetre portions of a 1% solution of sodium chloride were taken, to which were added 0·1 milligramme of copper and various amounts of lead. Sulphuretted hydrogen was then passed through the solution and from this stage the analysis was conducted according to the method of Bishop, with the exception of the final determination in which a modification was made, the reason for which will be given later.

The results obtained were as shown in Table V.

TABLE V.

Observation.	1	2	3	4	5	6
Lead added ..	0·00	0·00	0·01	0·02	0·04	0·06
Lead found ..	0·00	0·00	0·005	0·012	0·03	0·04

On account of what has been said above, this method is considered unsuitable when the amount of lead to be determined is less than 0·05 milligramme.

In addition to the points mentioned, two very grave errors have been introduced. I quote from the paper published in THE MEDICAL JOURNAL OF AUSTRALIA, April 21, 1928, page 484:

> In all estimations in this paper and those which follow, the lead concentration is always in the range of 0·1 milligramme to 0·2 milligramme. Thus when the lead in the unknown solution was expected to be less than 0·05 milligramme, sufficient standard solution was added to bring the lead value within the range given above, leading to an enhanced accuracy for the determination.

Also in the same publication is given a description of how the final determination is made:

> The comparison is then made with the following solution: Five cubic centimetres of one in two ammonium acetate solution, one cubic centimetre of 10% potassium cyanide solution, one cubic centimetre of 0·880 ammonium hydroxide, two drops of 1% gum arabic solution, two drops of sodium sulphide solution and the whole is made up to nine cubic centimetres with distilled water. This solution must be quite colourless. Sufficient of the lead acetate solution is then added from a burette until the colour developed is the same as that of the unknown solution. The amount of lead standard solution added is read off from the burette, which should be graduated to read 0·05 cubic centimetre. The solutions are then placed in the cups of a Duboscq colorimeter and the final comparison made.

The principle of adding lead to the unknown solution when lead is the substance to be determined cannot be condemned too strongly and particularly in this case where the method employed for the final determination is one which gives high results.

By the method of Bishop the colour in the unknown lead solution is produced by the addition of sodium sulphide whereby the lead present is precipitated as lead sulphide in a very finely divided condition. If the lead particles in this suspension were increased in size, an appreciable alteration in colour would take place, although the amount of lead present was the same. The standard with which the colour of this suspension is compared, is prepared, as stated above, by the addition of a standard lead solution to a solution which contains sodium sulphide; the result is that for small quantities of lead the colours produced in the unknown and standard suspensions are not comparable since they have been produced under different conditions. This is well shown in Table VI in which are compared the colours produced when a solution of sodium sulphide is added to a solution of lead and a solution of lead to a solution of sodium sulphide.

TABLE VI.

Lead present ..	0·00	0·02	0·04	0·06	0·08	0·10
Sulphide to lead..	0·00	0·02	0·04	0·06	0·08	0·10
Lead to sulphide..	0·00	0·015	0·03	0·04	0·06	0·08

Although the differences due to the method of determination may appear small, they are relatively very large when the amount of lead present in the sample is considered and may lead to an apparent lead content many times greater than that actually present, particularly when a large multiplication factor is used, as is done by Bishop.

In view of what has been said, it is of interest to compare the results obtained for the lead content of eggs determined:

1. By the method of Bishop;
2. By the method of Bishop modified so that the final determination was made without the addition of lead and the colours in both standard and unknown lead solutions produced in the same way.
3. By the method of Taylor.

The eggs taken were six new laid shop eggs. The yolks and whites weighed 293 grammes, these were well mixed and portions taken for analysis.

The results obtained by methods 1, 2, 3 were as shown in Table VII.

TABLE VII.

Method.	Amount of Wet Substance Taken in Grammes.	Total Lead Found in Milligrammes.	Net Lead Found in Milligrammes.	Lead, Milligrammes per 100 Grammes.
1	50	0·118	0·018	0·036
	10	0·118	0·018	0·18
	5	0·125	0·025	0·50
2	33	0·005	0·005	0·015
3	33	0·002	0·002	0·006

It will be seen from these figures that the lead content of 100 grammes of egg substance determined by the method of Bishop varies from 0·036 milligramme to 0·50 milligramme, according to the amount of egg substance taken.

This variation is due to the multiplication of the errors made in the final determinations, caused by the comparison of the colours of lead suspensions produced in different ways and accentuated by the

addition of 0·10 milligramme of lead to the solution, the lead content of which was to be determined.

The lead content determined by method 2 was 0·015 milligramme per 100 grammes and by method 3 0·006 milligramme per 100 grammes.

The figure by method 2 is slightly high, probably due to contamination during the drying process. That contamination did take place during drying is shown by the lead content of the wet substance and after it had dried at 105° C. for twenty-four and seventy-two hours respectively. These figures were obtained by method 3 and were 0·006, 0·014 and 0·03 milligramme per 100 grammes of wet substance, which shows that contamination to the extent of 0·024 milligramme of lead took place in three days.

In Table VIII the results are given of determinations carried out according to method 2 on the egg substance from six eggs obtained from Willoughby. The yolks and whites weighed 291 grammes and the analyses were made on the wet substance without preliminary drying.

TABLE VIII.

Yolk and Whites in Grammes.	Lead Added in Milligrammes.	Lead Found in Milligrammes.	Lead, Milligrammes per 100 Grammes.
97	0·00	0·01	0·01
97	0·02	0·03	0·01
97	0·02	0·03	0·01

These results agree very closely with those given in Table I and support the conclusion drawn that the lead content of a normal egg is not greater than 0·005 milligramme.

Summary.

The results given in this paper for the lead content of eggs obtained from various sources show that the amount of lead in an egg from a fowl which has been fed on ordinary food giving a lead intake of approximately 0·5 milligramme per day, does not exceed 0·005 milligramme.

A slight increase in this amount can be obtained by feeding a fowl on lead, but the increase is extremely small when compared with the amount of lead it is necessary for the fowl to ingest.

The lead content of a number of eggs has been determined by the method of Bishop and the errors in the method which gave rise to the high values found have been pointed out.

Determinations have also been made by the method of Bishop, modified so as to eliminate the errors mentioned and the results then obtained, agreed very closely with those obtained by my method.

Bishop and Cooksey in "An Additional Note" confirm the results of Bishop by a new method originally introduced by me. Since the errors in Bishop's analyses have been pointed out, it is obvious that errors must also exist in the new method as applied by Bishop and Cooksey to confirm the results of Bishop.

It would appear, therefore, highly probable that the high values for the lead contents of ordinary eggs given by Bishop and later confirmed by Bishop and Cooksey are due to the means employed to determine them.

ALBUMINURIA AND ECLAMPSIA.[1]

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HAVING been asked to read a paper before the Branch, my first difficulty was to find a subject which the audience would not know more about than I did myself and secondly to find an obstetric condition which is creating interest amongst the general body of medical men at the present time. My choice fell upon eclampsia and albuminuria which necessarily include pregnancy, toxæmia and nephritis.

I shall attempt to show you that though during the last twenty years very little progress has been made in determining the actual causation of these conditions, much attention has been paid of late to the subsequent history of patients, with the result that the opinion is developing that pregnancy is very detrimental if not distinctly injurious to a patient already suffering from chronic nephritis and also that the so-called toxæmia of pregnancy is directly harmful to the kidney and may eventually be followed by true nephritis.

Eclampsia is the term applied to a condition which develops in a woman during pregnancy or shortly after delivery. The clinical evidence of such a condition is the onset of fits or coma, usually preceded by a series of symptoms such as epigastric pain, headache, dimness of vision, œdema, raised blood pressure *et cetera* which syndrome is known as preeclampsia; in the great majority of cases albumin could have been detected in the urine for a longer or shorter period previous to the advent of serious symptoms.

Eclampsia has been known since the time of Hippocrates and has always created considerable interest in the medical profession, but unfortunately the actual causative agent is still unknown. Very little has been added to our knowledge during the last twenty years, in spite of a very large amount of study, observation and examination by representatives of every branch of medical science.

De Lee states that:

> In spite of the enormous amount of study that has been put upon it, the real cause of this affection is unknown. The theory that has the most plausibility is that eclampsism and eclampsia are one form of toxæmia, that a disturbance of metabolism occurs of physico-chemical nature, and that some noxa circulates in the blood, which produces the generalized hydrops, the liver changes, and directly or secondarily degenerative changes in the kidneys, and finally convulsions by direct toxic action on the anterior cerebral cortex.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on April 4, 1930.

I want you to notice that De Lee considers that this condition produces a degeneration of the kidney substance.

John S. Fairbairn, representing the Engish view of the subject, discusses eclampsia under the heading of "Toxæmia of Pregnancy" and states that:

> Although the pathological lesion and clinical manifestations justify the use of the term toxæmia, we are entirely ignorant of the nature of the intoxication, its origin, and even as to whether there is one, or more than one, toxic agent at work. There is a considerable body of evidence to show that the condition is due to protein intoxication; some would urge that it originates in the intestinal tract . . . some would give a prominent place to thyroid and parathyroid insufficiency, and to faults of the endocrine metabolism . . . the most generally accepted view is that the intoxication originates from the ovum, either the fœtus itself or its trophodermal elements.

In 1909 Eardley Holland published an exhaustive review of the investigations on this subject up to that time, and summarized as follows:

1. There is no special eclamptic poison.
2. The disease is an autointoxication, in which a profound disturbance of protein metabolism plays the chief part.
3. The chief toxic substances are the products of disintegration of protein.
4. These substances are produced by the raised activity of intracellular ferments, causing autolysis of the body cells.
5. The primary cause is to be sought in the placenta, which probably produces activating proteolytic ferments in other parts of the body.

I am quoting these authorities to show what little progress has been made during the last twenty years in the elucidation of the causation of toxæmia in pregnancy. Constant investigation has been carried on, but the actual cause of the eclamptic condition is still a matter of surmise. Certain hypotheses became popular for a period, but are not heard of much at the present time.

In the year 1903, Nicholson published a paper in the *Journal of Obstetrics and Gynæcology of the British Empire* on the cause of eclampsia being due to an insufficiency of iodothyrin, due to a want of the normal pregnancy hypertrophy of the thyreoid glands and for some years reports were published on the successful treatment of threatened eclampsia by the administration of thyreoid extract.

In De Lee's last edition this treatment is mentioned in a paragraph headed "Sundry Remedies Mentioned for the Sake of Completion."

Dr. R. H. Paramore published, in 1928, the result of some experiments he had made on intra-abdominal pressure embodied in a paper entitled "The Mechanistic Conception of Eclampsia." He made examinations of pregnant women with a mercurial manometer and a rubber bag in the rectum. He reported that during the later months of pregnancy, the pressure in the rectum became greater than the atmospheric pressure except in women with lax abdominal walls or when they were placed in the genu-pectoral position. He stated that this increased abdominal pressure was most evident in *primigravidæ* with tense abdominal walls and became more pronounced in patients with rapid enlargement of the uterus, such as occurred in hydramnios, multiple pregnancy and accidental hæmorrhage. Increase in the intraabdominal pressure would act detrimentally on the function of the liver and kidneys, by interfering with their blood supply, and albumin would soon appear in the urine.

I fail to see how this supposition explains all cases of eclampsia or albuminuria and I have not seen that it has been accepted, but it might explain the sudden appearance of albumin without other preeclamptic symptoms, which is sometimes seen in hydramnios and multiple pregnancy shortly before the onset of labour, the albumin disappearing very rapidly after delivery.

Nothing further has been discovered of late years to elucidate the actual causation of eclampsia and albuminuria in pregnancy, but a considerable amount of work has been carried out lately to demonstrate the harm that a prolonged toxæmia has upon the kidneys, whether previously affected with chronic nephritis or not.

James Young read a paper before the Royal Society of Medicine on November 16, 1928, in which he quoted figures showing that over 50% of patients who had been treated for eclampsia or albuminuria in a previous pregnancy, either had a recurrence of the toxæmia in a subsequent pregnancy or the pregnancy was interrupted by abortion or accidental hæmorrhage and at the conclusion of his paper, he stated that toxæmia of pregnancy can be regarded as the cause of all first attacks of pregnancy albuminuria, in previously healthy patients.

The renal damage in these patients is purely secondary to the toxæmia. As a result of this toxæmia a certain proportion, about 10%, develop frank chronic nephritis whether or not they become pregnant again, a further proportion, about 40%, recover their kidney function completely after delivery and subsequently may have normal pregnancies without albuminuria. The remainder, about 50%, recover their kidney function, only so far as to be free from the signs of renal disease in the intervals between pregnancies. If they subsequently become pregnant, they suffer with recurrent albuminuria. Recurrent albuminuria may be looked upon as due partly to a subliminal pregnancy toxæmia and partly to a preexisting occult nephritis caused by the first toxæmic pregnancy. Pregnancy, says Young, is the most delicate test of renal function we possess.

You will realize that investigations of this sort take years to produce, as the difficulty of following up hospital patients is very great. I have commenced to investigate in a small way and to follow up the material that is available in Brisbane and to commence with the incidence of albuminuria is higher in Brisbane than in other parts of the world; from reports sent in by members of the Obstetric section (all private patients) albumin was reported in the last two reports for the years 1928 and 1929

respectively, as 79‰ and 75‰. In the 1928 New Zealand report, albumin is recorded as 13‰. De Lee puts albumin down as 30‰ to 50‰ and Fairbairn as 50‰.

Thirty-nine patients, confined in the Lady Bowen Hospital since July, 1928, who had given evidence of toxæmia have been followed up and as a result of these investigations, have been divided into three groups:

1. Definite nephritics. Eight have been placed in this group. The infant mortality was five or 62·4%. De Lees gives the figures as 60%. Two of the mothers died within three months of the confinement from conditions associated with nephritis, namely uræmia and heart failure. This is a maternal death rate of 25%. De Lees quotes a death rate of 30%.

2. Patients with pure toxæmia. Seventeen have been placed in this group with no maternal or infantile deaths.

3. Unclassified patients but still under observation; this group is divided into two classes. (*a*) Probable nephritics. Six have been placed in this group, four of the mothers were delivered of stillborn children, two of which were macerated. (*b*) Probable toxæmics. Nine have been placed in this series, with an infant mortality of two.

I submit examples of each group to show what investigations are being made. I fully realize that this series of thirty-nine cases is of no value from a statistical point of view, but they give a local interest and bear out the opinions quoted in the paper.

Patients with a Diagnosis of Definite Nephritis.

CASE I.—A.S., pregnant for the fifth time, was thirty-one years of age. She was admitted on April 7, 1929. She gave the history that her first and second confinements had been normal. In the third pregnancy surgical induction had been carried out for severe preeclamptic symptoms. Eclampsia had occurred in the fourth pregnancy. The patient's blood urea content was 23 milligrammes per hundred cubic centimetres. The figures obtained by the urea concentration test were 1·7, 0·8 and 2·7. The systolic blood pressure was 210 millimetres of mercury. In the Mosenthal test the amount of day urine was 2,400 cubic centimetres (eighty fluid ounces), the specific gravity was 1007; the night urine was 210 cubic centimetres (seven fluid ounces) and the specific gravity was 1015. Owing to the increase of preeclamptic symptoms labour was induced in the twenty-seventh week and a dead child was born.

On April 19, 1929, the blood urea was 38 milligrammes *per centum* and the figures obtained by the urea concentration test were 0·5, 0·7 and 0·7.

On July 20, 1929, the blood urea was 38 milligrammes *per centum*, the figures obtained by the urea concentration test were 0·8 and 1·8, there was "one quarter" albumin present, the systolic blood pressure was 190 millimetres and the diastolic pressure 140 millimetres of mercury.

This patient's condition was diagnosed as chronic nephritis; there was an increasing blood urea content, with persistent albuminuria and high blood pressure.

CASE II.—M.I., pregnant for the first time, was twenty-eight years of age. She was admitted in April, 1929. She was supposed to have had lead poisoning when a child with paralysis. The blood urea was 53 milligrammes *per centum*. The figures obtained by the urea concentration test were 0·5, 0·6, 0·8. In the Mosenthal test the day urine was 630 cubic centimetres (21 fluid ounces) and the specific gravity was 1011; the night urine was 1,140 cubic centimetres (38 fluid ounces) and the specific gravity was 1008. The systolic blood pressure was 130 and the diastolic pressure 86 millimetres of mercury. The albumin was "one third."

On April 27, 1929, labour was induced by surgical means in the thirty-fifth week and a dead child was born.

On May 1, 1929, the systolic blood pressure was 152 and the diastolic pressure 120 millimetres of mercury.

On October 22, 1929, the systolic blood pressure was 128 and the diastolic pressure 80 millimetres of mercury. The blood urea was 66 milligrammes *per centum* and the figures obtained with the urea concentration test were 0·5, 0·7 and 1·2. There was a cloud of albumin and the serum did not react to the Wassermann test.

CASE III.—M.M., aged twenty-five years, was pregnant for the first time. She was attending the medical outpatient department for kidney trouble and was found to be seven weeks pregnant.

On August 23, 1929, the blood urea was 53 milligrammes *per centum*. The figures obtained by the urea concentration test were 1·1, 1·8 and 0·9. The systolic blood pressure was 165 and the diastolic pressure 115 millimetres of mercury. The urine contained a cloud of albumin. After one week's rest in bed, on August 31, 1929, the blood urea was 52 milligrammes *per centum*. The figures obtained by the urea concentration test were 2·7, 2·0 and 2·3. The systolic blood pressure was 160 and the diastolic pressure 100 millimetres of mercury. The urine contained a cloud of albumin.

On October 22, 1929, the blood urea was 53 milligrammes *per centum*. The figures obtained by the urea concentration test were 2·9, 2·0 and 2·3. The systolic blood pressure was 170 and the diastolic pressure 110 millimetres of mercury. In the Mosenthal test the average specific gravity of the urine day and night was 1010.

On November 5, 1929, the figures from the urea concentration test were 2·1, 1·1 and 2·9. The urine had a constant low specific gravity of under 1010. The systolic blood pressure was 155 and the diastolic pressure 100.

On December 10, 1929, the urine contained a cloud of albumin. The systolic blood pressure was 172 and the diastolic pressure was 120 millimetres of mercury.

On December 27, 1929, the urine contained a cloud of albumin. The systolic blood pressure was 198 and the diastolic pressure 130 millimetres of mercury.

On December 22, 1929, the preeclamptic symptoms increased, the patient complained of severe headaches and facial paralysis was present. Labour was induced by surgical means and a macerated fœtus in the twenty-ninth week of its development and weighing 3·1 kilograms (two pounds eleven ounces) was delivered.

On January 3, 1930, the urine contained a cloud of albumin. The systolic blood pressure was 190 and the diastolic pressure 120 millimetres of mercury. The patient was suffering from chronic nephritis and the detrimental influence that a twenty-seven weeks' pregnancy had on the condition was manifest.

CASE IV.—M.M., aged 35 years, was pregnant for the fourth time. The first child had been born fourteen years before and the second twelve years before. Both these children are well and healthy. During the third pregnancy albumin had been present in the urine and spontaneous premature labour had occurred at five and a half months.

On October 22, 1929, the patient had been pregnant twenty-nine weeks. The specific gravity of the urine was 1014; it contained "half" albumin. The systolic blood pressure was 220 and the diastolic pressure 140 millimetres of mercury. The blood urea was 35 milligrammes *per centum*. The figures obtained by the urea concentration test were 0·9, 0·6 and 1·3.

On October 29, 1929, there was an increase of preeclamptic symptoms. Labour was induced and a live child weighing 1·0 kilogram (two pounds five ounces) was delivered; it died a few hours after birth.

On March 3, 1930, four months after confinement, the systolic blood pressure was 236 and the diastolic pressure 140 millimetres of mercury. The urine contained a cloud of albumin; its specific gravity was 1004.

This case is an example of the toxæmia in the third pregnancy being repeated in the fourth, the kidney becoming permanently damaged.

CASE V.—H. E. McN., aged forty-four years, was pregnant for the ninth time. The patient's blood did not react to the Wassermann test. The first two children were stillborn. Five were born alive. The patient suffered from "kidney trouble" during her eighth pregnancy. The systolic blood pressure was 170 millimetres of mercury. The blood urea was 69 milligrammes *per centum*. The figures obtained by the urea concentration test were 1·7 and 2·8.

On September 21, 1929, during the thirty-seventh week of pregnancy, the blood urea was 23 milligrammes *per centum* and the figures obtained by the urea concentration test were 1·2, 0·7 and 1·6. The urine contained a cloud of albumin. The systolic blood pressure was 200 and the diastolic pressure 120 millimetres of mercury. In the Mosenthal test the day urine amounted to 450 cubic centimetres (fifteen fluid ounces), its specific gravity was 1008; the night urine amounted to 600 cubic centimetres (twenty fluid ounces), its specific gravity was 1014.

On October 2, 1929, labour was induced by surgical means and a dead child was born.

On February 26, 1930, four and a half months after the termination of the pregnancy, the systolic blood pressure was 186 and the diastolic pressure 100 millimetres of mercury. The urine contained a cloud of albumin.

CASE VI.—A.G., aged forty-two years, was pregnant for the third time.

On November 5, 1929, the urine contained "a third" albumin. The systolic blood pressure was 230 and the diastolic pressure 125 millimetres of mercury. The blood urea was 160 milligrammes *per centum*.

On November 10, 1929, natural delivery occurred at approximately the thirty-seventh week, the child being alive and weighing 1·36 kilograms (three pounds ten ounces).

On November 25, 1929, the patient was transferred to the General Hospital. The blood urea was 145 milligrammes *per centum*. The figures obtained by the urea concentration test were 0·7, 0·7 and 0·8.

On March 28, 1930, the blood urea was 190 milligrammes *per centum*. The figures obtained by the urea concentration test were 1·1, 0·1 and 0·1. The patient remained in hospital for four months and died a few weeks after leaving.

Patients Placed in the Toxæmic Group.

CASE VII.—T.T. was pregnant for the first time.

On March 1, 1929, the patient was admitted in a fit, having three fits in all. Natural delivery occurred, the child being alive. The blood urea was 33 milligrammes *per centum*. In the Mosenthal test the day urine amounted to 990 cubic centimetres (thirty-three fluid ounces), its specific gravity was 1012; the night urine amounted to 1,140 cubic centimetres (thirty-eight fluid ounces), its specific gravity was 1010.

On May 2, 1929, the blood urea was 25 milligrammes *per centum*. The figure obtained by the urea concentration test was 2·5. The systolic blood pressure was 125 and the diastolic pressure 75 millimetres of mercury.

CASE VIII.—C.A.B., aged 44 years, was pregnant for the seventh time. All previous confinements had been normal.

On April 16, 1929, the patient was admitted in a fit, having had twenty-five fits altogether. The child was born alive, weighing six pounds eight ounces. In the Mosenthal test the average specific gravity of the day urine was 1009 and of the night urine 1010.

On February 16, 1930, the specific gravity of the urine was 1020; it contained no albumin. The blood urea was 33 milligrammes *per centum* and the figures obtained by the urea concentration test were 1·9, 3·1 and 3·1. The systolic blood pressure was 138 and the diastolic pressure 90 millimetres of mercury.

Patients Under Observation.

The following are examples of patients under observation who will probably eventually be placed in the nephritic group:

CASE IX.—I.S., aged thirty-eight years, was pregnant for the sixth time. She had had five healthy children and had had no trouble in any previous confinement and no previous illnesses. As a result of antenatal examination the blood urea was found to be 65 milligrammes *per centum*. The figures obtained by the urea concentration test were 0·7, 1·0 and 1·0. The systolic blood pressure was 190 millimetres of mercury. In the Mosenthal test the day urine amounted to 1,950 cubic centimetres (65 fluid ounces), its specific gravity being 1002; the night urine amounted to 1,560 cubic centimetres (52 fluid ounces), its specific gravity being 1004. Spontaneous delivery of a macerated fœtus took place at approximately the twenty-fourth week.

CASE X.—S.J., aged thirty-seven years, was pregnant for the first time and had had no previous illnesses.

On October 21, 1929, the urine contained "a quarter" albumin and had a specific gravity of 1010. The systolic blood pressure was 176 and the diastolic pressure 120 millimetres of mercury.

On November 4, 1929, the urine contained "a quarter" albumin and had a specific gravity of 1010. The systolic blood pressure was 180 and the diastolic pressure 110 millimetres of mercury.

On November 5, 1929, the blood urea was 57 milligrammes *per centum* and the figures obtained by the urea concentration test were 2·4, 2·4 and 2·5.

On November 13, 1929, eclamptic convulsions occurred and labour was induced at approximately the thirty-seventh week. The child was still-born.

On November 21, 1929, in the Mosenthal test the day urine amounted to 780 cubic centimetres (26 fluid ounces), its specific gravity being 1005; the night urine amounted to 570 cubic centimetres (19 fluid ounces), its specific gravity being 1010.

On November 25, 1929, the systolic blood pressure was 160 and the diastolic pressure 90 millimetres of mercury. The urine contained a cloud of albumin.

On February 13, 1930, two and a half months after delivery the systolic blood pressure was 170 and the diastolic pressure 90 millimetres of mercury. There was no albumin in the urine which had a specific gravity of 1016. The blood urea was 75 milligrammes *per centum* and the figures obtained in the urea concentration test were 1·2, 1·3 and 2·1.

The following two records were obtained from the General Hospital and they suggest that, if post-natal care had been taken, the nephritic condition following the toxæmic pregnancy might have been modified:

CASE XI.—T.A.T., aged 45 years, had had no previous illnesses. She had had two children, the last being born six years before. Severe albuminuria had occurred during the greater part of the last pregnancy. The patient had had one miscarriage after the last child was born.

On September 14, 1929, the patient was admitted to the General Hospital with uræmia. The blood urea was 400 milligrammes *per centum*. The patient died the next day.

CASE XII.—E.L., aged thirty-eight years, had had three children, the youngest being six years of age. Since the birth of the last child she had had three miscarriages and one premature labour.

On December 27, 1929, after a pregnancy of three and a half months, the blood urea was 89 milligrammes *per centum* and the figures obtained by the urea concentration test were 1·0, 1·0, 1·0. The systolic blood pressure was 235 and the diastolic pressure 140 millimetres of mercury.

On January 10, 1930, the blood urea was 300 milligrammes *per centum*. The uterus had been emptied on January 6, 1930. The patient died on January 14, 1930.

Prophylaxis.

I do not intend to discuss the ordinary treatment of albuminuria and eclampsia, but I should like to raise a discussion on the correct prophylactic treatment, first of all, of the true nephritics and secondly of those patients who have suffered from a prolonged albuminuria or a toxæmic eclampsia in any one of their previous pregnancies.

There is a consensus of opinion as to the danger of allowing a pregnancy to continue in a woman suffering from chronic nephritis, but all the same one constantly sees these patients being allowed to go through their pregnancy and we seldom hear of efficient steps being taken to prevent future pregnancies.

It is accepted that pregnancy is very detrimental to the function of a kidney affected with chronic interstitial nephritis, that the extent of the injury is almost directly proportional to the length of time the pregnancy has been allowed to exist.

It is also recognized that the mother may at any time develop convulsions or retinitis which may come on suddenly and result in permanent disablement of vision; if she has not been relieved by an abortion or premature labour, which according to De Lee will occur in about 66% of cases, she will reach full term, to be delivered of a macerated or still-born fœtus (60%) and risk a maternal mortality of 30% from eclampsia, *post partum* hæmorrhage and puerperal sepsis. Live children born in these circumstances are notably badly developed and according to the records of the Glasgow Royal Maternity Hospital, reported in the "Medical Annual" of 1929: "The mortality of these children dying during the puerperium, is three times as great as that of ordinary children."

Beaumont and Dodds state that with a blood urea and non-protein nitrogen of 40 milligrammes *per centum* the pregnancy should be terminated, as, although it is possible for such pregnancies to go to term, experience has shown that their renal condition is much worse after delivery than before.

In 1907, Professor J. Whitridge Williams and J. Morris Slemons made the statement that:

> Opinion is developing which favours an early sacrifice of the fœtus rather than hazarding the life of the mother by undue delay.

But though the opinion was developing in 1907, it has by no means matured in twenty-three years, nor is it definitely settled that once a patient is considered to be suffering from chronic nephritis, the correct prophylactic treatment is to bring about an occlusion of the Fallopian tubes by surgical methods.

The other group of patients, apparently free from nephritis to begin with, but developing toxæmia in one of their pregnancies, was supposed to be quite distinct from the nephritic variety, but the opinion is developing more and more that such is not the case and it is now believed that the toxæmia of pregnancy shown by eclampsia and albuminuria has a decidedly detrimental effect on the kidneys.

James Young in November, 1928, and Gibberd in April, 1929, both published articles showing that 10% of all women passing through a toxæmic pregnancy did not recover when the pregnancy terminated, but immediately showed signs of chronic nephritis; that 50%, though recovering to a certain extent after the pregnancy was over, showed signs of toxæmia in subsequent pregnancies and that apparently only 40% escaped without some permanent injury to the kidney.

Peckham published a paper, September, 1929, giving the results of investigations made at the Johns Hopkins Hospital. Since 1919 he has made arrangements to obtain a reexamination of all patients showing toxæmic symptoms during their pregnancy; he makes this examination thirteen months after labour has terminated. He quotes a record of 77 cases and shows a nephritic incidence of 22·1%. These figures are higher than those of Young and Gibberd, because the patients suffering from a preexisting nephritis have not been separated. Peckham publishes the following interesting summary: Nephritis is more likely to occur after severe than mild eclampsia, the severity is not estimated by the number of fits, but by the length of time the toxæmia has existed. The percentage incidence of nephritis increases with the age of the patient, also that multiparous patients are more prone to be affected than primiparous. He considers that the impairment of the kidney depends directly on the amount of hypertension and albumin present. With a blood pressure of 200 millimetres of mercury or more and albumin of at least 10 grammes, three-quarters of these patients develop nephritis.

He considers also that a slow return of the blood pressure to normal and the persistence of albuminuria, is of great prognostic importance. The report of the patients I have followed up, 39 in number, shows a percentage of 20·5, but so far I have not been able to separate those that were suffering from nephritis before the pregnancy commenced. We suspect that four have been suffering from chronic nephritis, one is doubtful, the other three have experienced two and one three previous toxæmic pregnancies. In my opinion these patients should have the opportunity of being sterilized.

In working out the age incidence of my series, the results show a steady increase with age.

Up to 19 years of age 0% developed nephritis, from 20 to 29 years of age 9·5% developed nephritis, from 30 to 39 years of age 33·3% developed nephritis, from 40 years of age onwards 50% developed nephritis.

In regard to blood pressure of patients with a systolic blood pressure of 70 to 170 millimetres of mercury, 15% developed nephritis; of those with a systolic blood pressure of 170 to 200 millimetres of mercury, 18% developed nephritis; of those with a systolic blood pressure of 200 millimetres and over, 75% developed nephritis.

In conclusion I wish to emphasize the serious effect that results from allowing nephritic women

to become pregnant and, if they become pregnant, in allowing the pregnancy to continue. I also wish to suggest that every case of pregnancy toxæmia requires very careful consideration, remembering that only 40% of women who pass through a toxæmic pregnancy escape damage to their kidneys.

Young in his paper of November, 1928, states that in his opinion no toxæmic patient seen before the seventh month, except those with the very mildest conditions, should be kept under treatment for more than two weeks and then only if the progress is good, especially so if the woman has suffered damage in her previous pregnancies. He also states that no woman should be exposed to further risk if she has had two or more toxic pregnancies, for the risk of future recurrence rises rapidly with the number of pregnancies so vitiated.

If the line of prophylactic treatment, suggested above, were to be carried out, the maternal and infantile mortality tables would be improved and the prolongation of the mother's life, during the important period when she is rearing a family, would be of considerable economic value to the State.

Thanks.

I wish to express my thanks to the members of the Lady Bowen Hospital staff for allowing me to make use of their case records, to the out-patient physicians of the Brisbane General Hospital for checking my post-natal examinations and to Dr. Duhig and his staff for the bio-chemical reports.

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Reports of Cases.

THE USE OF CHROMIC ACID IN AURAL SUPPURATION.

By T. S. KIRKLAND, M.D., F.R.C.S. (Edin.),
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I HAVE used chromic acid frequently for many years. It is particularly useful in suppuration of the middle ear when there is no involvement of the attic or mastoid antrum. An application may be made to the inner wall of the middle ear, especially if it presents a granular or polypoid condition. It is beautiful to see how it promotes epithelialization.

In a case recently treated by me, where the drum was entirely destroyed with no sign of the ossicles and in which there was not a vestige of epithelium to be seen, there developed a complete new epithelial covering of the inner wall in six months' time.

The discharge in this case was muco-purulent and the mucous membrane of the inner wall appeared congested and red with no granulations. An application of a 20% solution of chromic acid to the inner wall intended to convert this into a non-secreting surface with the result that a total new membrane grew up and covered over the secreting surface.

There is now no discharge and I regard the patient as cured for all time.

The first case in which I tried this form of treatment in 1897 was one of chronic suppuration of many years' standing and the patient had been treated by several men without any effect. The hypotympanic space was deeper than usual and granular. A tiny bead of chromic acid fused on a probe and subsequently neutralized by an alkaline lotion in a short time enabled the epithelium to grow and within three months the whole surface was dry and healed over. It is the continual bathing of the mucous surface with germ life that prevents this form of cure. Recently in a case of acute suppuration of the mastoid in a patient with chronic Bright's disease whose profound pallor was secondary to this condition, there was no sign of healing. The wound discharged blood and pus and seemed to require curetting. However, I passed every day a probe armed with cotton wool and dipped into a solution of 1% chromic acid. The change was astounding and in a short time complete healing occurred.

I make this short communication in the hope it may help in cases of a similar nature. Chromic acid is a powerful germicide and, handled carefully in proper strength to suit the condition, must prove valuable.

No doubt one has a disinclination to use such a powerful drug in a delicate place like the ear, but there is really no danger of doing harm.

Reviews.

THE TECHNIQUE OF LOCAL ANÆSTHESIA.

"PRACTICAL LOCAL ANESTHESIA AND ITS SURGICAL TECHNIC," by Robert Emmett Farr, was first published six years ago. The second edition has been revised and enlarged and contains more illustrations.[1] There are fewer photographs of actual operations, as it is considered that there is now little need to advertise the merits of local anæsthesia by pictures showing the patient's smiling face barely screened from gaping operation wounds. Advantage has been taken to increase the number of pictures of the technique. These are of the high standard usual in American publications, but it is irritating to find them referred to in the text by wrong number of page or figure. The index is rather limited and would be improved by more cross references.

Infiltration and infiltration block are preferred to regional anæsthesia in fields in which the latter is not definitely indicated. Sacral anæsthesia is advocated for many conditions; and experiments on the cadaver have led to the use of more concentrated solutions, less than thirty cubic centimetres of a 2% or 3% solution being employed. Labat and Hertzler, on the other hand, use larger amounts of 1% solution. Spinal anæsthesia is only just mentioned, the sole index reference is to intraspinal anæsthesia which appears as one line on page 125. There is no account of Pitkin's controllable spinal anæsthesia.

In practised hands the limits of local anæsthesia are considered to be few and come under two classes. First

[1] "Practical Local Anæsthesia and its Surgical Technic," by R. E. Farr, M.D., F.A.C.S.; Second Edition, thoroughly revised; 1929. Philadelphia: Lea and Febiger. Royal 8vo., pp. 611, with illustrations. Price $9.00 net.

there is the patient who is incompatible from temperament or youth. It is not surprising that the youngest patient operated upon for cleft palate was fifteen years old and that ether is suggested for this type of operation in children. Secondly, certain infective conditions are unsuitable and acute mastoid disease and pelvic abscess are mentioned as conditions in which the injections might cause spread of the disease. The danger of this procedure has recently been pointed out by Professor J. Vegrassat (*Revue de Stomatologie*, July, 1929, page 411) who quotes cases of septicæmia and death following local anæsthesia in septic conditions.

Excision of varicose veins under local anæsthesia is described and two illustrations are given, but no mention is made of the safer and simpler method of cure by injection.

The subject of narcotics is discussed at length and excessive doses are condemned. Gwathmey's claims for the synergistic effect of magnesium sulphate and morphine were investigated for two years and though the effect of the morphine was somewhat enhanced, it was not found to be as great as Gwathmey suggests. Also a number of sloughs occurred where the solution was injected intradermally and subdermally. "Avertin" and the barbituric salts receive no mention. Ethylene is recommended when general anæsthesia is required to reinforce local injections.

Farr uses a special injector driven by an electric motor and controlled by a cut-off with a pistol grip. The advantages claimed for this instrument are that it is a better means of controlling the needle, gives a constant supply of solution without removing the eyes from the field of operation, produces a quicker induction, eliminates "muscle tire" and that the cost of upkeep is less than with syringes. This machine is set up and kept in order by the psycho-anæsthetist whose characters and duties are set forth at length. "This individual should by preference be a woman, and a tactful trained nurse who is well trained in the administration of general anesthesia." Her duties are many and varied and include care of the patient, supervision of the machinery, recording pulse rate and blood pressures, attention to the ventilation of the theatre, informing the surgeon of the amount of solution used and if general anæsthesia is required and, lastly, working the "Victrola" to supply suitable accompanying music.

The details of technique and the author's special instruments are very thoroughly described and many valuable suggestions are made. The subdermal method of anæsthetizing the skin avoids the multiple puncturing of the skin, a common and objectionable feature so frequently seen in local anæsthesia. Suction replaces sponging almost entirely and so lessens the hæmorrhage. It is pointed out that small vessels cease to bleed quickly through the formation of clots in their severed ends, provided the clot is not forcibly removed by sponging. For the skin incision it is possible to avoid pressure by retracting the skin vertically between two towel clips. Artificial illumination is depended upon entirely and a complicated operating lamp is described which combines six incandescent bulbs of "75 to 100 watts each." These globes are fixed some fifteen centimetres (six inches) above the head of the operator; a motor and suction fan are required to reduce the heat. The "Scialitic" type of lamp would appear to have many advantages over this arrangement and the upkeep cost would be much less.

The most interesting and instructive part of the book is that upon strategy. Here are included many details that are apt to be forgotten by those operating on an insensible patient. Farr considers the comfort of the patient as one of the most essential adjuncts to successful local anæsthesia. Worry, fuss and excessive preparation are condemned. Narcotics are used to prevent the preoperative distress rather than to help poor anæsthesia. The patient is gently moved on a special lifter from his bed to an efficiently padded table which is provided with comfortable rests for the arms and legs. Yet there are still surgeons who refuse to give their patients any drug to alleviate the horrors of the waiting time, let them walk to the theatre and lie down on a hard metal table which is scarcely insulated by a folded blanket. If they complain of the hard iron kidney support, they are told that they will soon be asleep; and their subsequent pain in the back is treated with morphine or scepticism according to the humanity of the surgeon.

This book may be read by all surgeons and anæsthetists with advantage to themselves and the great benefit of their patients.

NEUROSIS.

IN an introduction Dr. Cecil Webb-Johnson states that his volume on "Nerve Troubles, Causes and Cures" is designed to "offer a few useful hints to the neurasthenic, warning them of what to avoid and advising on methods of restoring vitality to the shattered nervous system."[1]

The author has written works on many topics, including dietaries for various ages, obesity, twilight sleep, women and beauty *et cetera*. His latest under discussion may possibly have a vogue with the lay public, but will not be prescribed as a reader by physicians.

One of the bugbears in treating neurotics is their firm belief in heredity and inborn nervous weakness. Dr. Webb-Johnson strengthens this by a chapter on heredity and another on children in which he lays stress on the alleged nervousness of those born in the war years. Only in a last brief sentence does he gild the pill by a statement that the sufferer can be cured of his neurasthenia, whether inherited or acquired.

It is probable that a psychopath will receive knowledge of additional symptoms for his armoury by perusing the account of various obsessions, phobias, hysterias *et cetera* mentioned in the chapters on nervous doubts and fears, hysteria and control of the emotions.

Perhaps the worst feature in the book is the manner in which drugs and stimulants are decried. We are reminded that tea drinking leads to insomnia, that General Grant died from cancer of the throat produced by cigar smoking, that veronal, chloral and sulphonal quickly obtain a hold on the victim and set up a craving *et cetera*.

Whilst such doctrines may be partially true in theory, their adoption in practice would serve as harmful suggestions.

Though there is an undoubted need for a small book on nerves for the modern layman, it must be planned on different lines from those of the volume on review.

PHARMACOLOGY.

THE new seventh edition of Professor Dixon's "Manual of Pharmacology" has been received.[2] This popular work with its clear and concise treatment of its subject and its profusion of diagrams has now the hall-mark of a classic. The present edition has been completely revised and brought up to date. The book is too well known to need any detailed introduction. Taken as a standard modern text book, this manual illustrates the difficulty which still confronts the pharmacologist in treating systematically the miscellaneous elements of the pharmacopœia. Those who were introduced to *materia medica* in the early years of the present century or before, will remember that the drugs were classified under two main groups according to their derivation from the vegetable or the animal kingdom, the former group being subdivided on the basis of botanical families. Professor Dixon presents a grouping according to pharmacological affinities, with the further deciding factor of chemical relationship intruding now and again, for example, in the group of the halogens. Ergot and cinchona are isolated in chapters to themselves. A wraith of the animal kingdom division still holds sufficient force to divorce adrenalin from the group of drugs acting on nerve endings. This leads to an anomaly, as adrenalin's affinity, ephedrine, thereby is grouped under the heading

[1] "Nerve Troubles, Causes and Cures," by Cecil Webb-Johnson, M.B., Ch.B.; 1929. London: Methuen and Company, Limited. Crown 8vo., pp. 102. Price: 2s. 6d. net.

[2] "A Manual of Pharmacology," by Walter E. Dixon, M.A., M.D., B.S., B.Sc., D.P.H., F.R.S.; Seventh Edition; 1929. London: Edward Arnold and Company. Demy 8vo., pp. 496, with illustrations. Price: 18s. net.

of animal extracts. A slightly more explicit exposition of the dual innervation of the involuntary muscular and glandular system and a more complete grouping of active principles according to their actions there would add to the value of the facts presented fully, but in different sections of the book. One slight inaccuracy was noted in regard to the induction time and duration of anæsthesia by ethyl chloride. Also an unsophisticated reader might draw the deduction from the article on cocaine that ten grains represent a minimal fatal dose. These are, however, but carping criticisms of an excellent book.

AN INTRODUCTION TO BACTERIOLOGY.

THE third edition of the "Handbook of Bacteriology for Nurses," by Dr. Harry W. Carey, of New York, is noteworthy for the amount of detail considered appropriate to the title of the work.[1] The author has sought to justify this fullness of treatment on the grounds that the requirements in America of the National League for Nursing and of the State Boards of Education are more exacting than formerly. The lengthy and strenuous practical training undergone by nurses in Australia would appear to leave little opportunity for the amassing of so great an amount of technical and general knowledge in bacteriology. It is improbable that any nurse, unless training as a technical assistant in a laboratory, would make subsequent practical use of information regarding complement fixation, allergy and anaphylaxis and similar subjects or concerning the bacillus of rhinoscleroma, Ducrey's bacillus or the *Spirochæta pertenuis*, to mention a few of the less known organisms described in the book.

Regarded, however, as a text book for the scientific student entering upon a course of bacteriology, it is written clearly and with interest and is well illustrated. Well known organisms are described in their appropriate groups and a simple account is given of the protozoa and higher bacteria. Diseases caused by these organisms are described and newer work in treatment is mentioned, especially that in connexion with infectious diseases. The technique and advantages of the Schick and Dick tests and the serum treatment of measles are dealt with adequately. We cannot, however, agree with the statement that measles is spread by "attendants on the patient, carpets, furniture and flies." At the end of each chapter is a comprehensive series of questions and the book ends with a number of laboratory exercises and demonstrations and a glossary of scientific terms.

Analytical Department.

"LISTERINE."

THE Lambert Pharmacal Company (Australia), Limited, has recently forwarded to us samples of "Listerine."

"Listerine" is a well known mild antiseptic solution. It is essentially a saturated solution of boric acid to which have been added benzoic acid and the active principles of thyme, eucalyptus, gaultheria, mentha and baptisia. It is a pleasant smelling, pleasant tasting fluid with a slightly acid reaction. It is manufactured by the Lambert Pharmacal Company (Australia), Limited, of Mountain Street, Sydney.

The factory premises are light and airy and very clean. The work people, too, are clean in dress and appearance.

The various ingredients, which are of high quality, are placed in a vat and continuously stirred for some hours. The solution is then allowed to stand for some time and then filtered. It is bottled in an automatic filling machine. Great care seems to be taken throughout to insure a good product.

Examination by our analyst showed that "Listerine" is a weak antiseptic fluid. It has a carbolic coefficient of about 0·05, but the undiluted fluid kills organisms rapidly. As it is recommended to use the fluid for most purposes, such as mouth wash, gargle *et cetera* in undiluted form it has sufficient antiseptic strength to destroy large numbers of bacteria after very short exposure and apparently without harm to epithelial surfaces.

"Listerine" can be recommended as a pleasant and efficient mild antiseptic solution.

"LISTERINE" TOOTH PASTE.

"LISTERINE" tooth paste is manufactured by the Lambert Pharmacal Company (Australia), Limited, in the same premises as "Listerine." The abrasive materials are calcium sulphate and calcium phosphate and there is no chalk nor soap. Practically the same essential oils are used for flavouring this tooth paste as are used in "Listerine."

In the manufacture of "Listerine" tooth paste modern machinery is used. The powders are mixed and sieved through fine meshed sieves in a closed machine. The mixed powders with glycerin and other fluid ingredients are mixed in a mixing machine until a thoroughly homogeneous paste is formed. The paste then passes through a chute to an automatic filling machine where the tin tubes are filled. At no stage of the process is there any handling and everything is kept very clean.

Our analyst reports that "Listerine" tooth paste is one of the modern acid soapless tooth pastes. The individual ingredients are of the highest quality. The abrasive materials consist of very small broken crystals and they will not scratch glass. The paste is distinctly acid in reaction having a pH of about 5. The manufacturers do not claim any antiseptic action for the tooth paste, but the essential oils added should make it mildly antiseptic.

"Listerine" tooth paste is a very fine example of an acid, non-soapy tooth paste.

"QUINBY."

"QUINBY" is the name given to a preparation of iodobismuthate of quinine. It was first introduced by Fournier and Guenot to the *Société française de Dermatologie et de Syphiligraphie* in 1921. It is claimed to be a specific for syphilis in all stages of the disease and the beneficial reaction is held to be the result of the combined action of iodide of quinine and of the spirillicide, bismuth. Reports of successful treatment by "Quinby" have appeared in many medical journals.

At our request several medical practitioners, specializing in the study and treatment of venereal disease, have used "Quinby" in their practices and have forwarded details of their results and expressions of their opinion of its value. Without exception the reports are favourable. In one series twenty-five patients were treated. Seven of them were suffering from cerebro-spinal syphilis. Two were intolerant of arsenic and manifested a mild dermatitis; they also manifested relapsing signs of syphilis while under treatment with another bismuth preparation. When given "Quinby" all these patients in the series made rapid progress. None of the medical practitioners in question has reported either clinical or serological relapse. There have been no "toxic" effects.

"Quinby" is produced in two forms—one, "soluble" in bi-distilled water and the other suspended in oil. One practitioner reported that some patients had experienced pain at the site of injection of "Quinby" "soluble." None of the other practitioners had had this experience. Several reported particularly on its effect on the response to the Wassermann test in tertiary syphilis and others stated that it was very satisfactory in syphilis of the central nervous system. It may be concluded that "Quinby" is a well-prepared and potent form of bismuth, suitable for use in all stages of syphilis. It has often produced satisfactory results when other antisyphilitic treatment has failed. We have yet to hear a well founded adverse opinion of its therapeutic value.

[1] "Handbook of Bacteriology for Nurses," by Harry W. Carey, A.B., M.D.; Third Revised and Enlarged Edition; 1930. Philadelphia: F. A. Davis Company. Demy 8vo., pp. 282, with illustrations. Price: $2·25 net.

The Medical Journal of Australia

SATURDAY, MAY 24, 1930.

Experimental Medicine.

THE report of the Medical Research Council always contains much that is of vital importance to both medical practitioners engaged in private and hospital practice and those whose activities are devoted to research. The report for the year 1928-1929, just received, is no exception to the rule; it has the added interest of containing a review of the past five years' work and a discussion of the outlook for the future. The report is divided into eight parts covering all the activities of the Council. It is proposed in the present instance to make reference to only one section of the introduction, that dealing with the relationship between clinical research and experimental medicine. It is necessary before considering this aspect, to give some indication of the scope of the work undertaken by the Council during the year. This is perhaps best indicated by drawing attention to the general use made of the available funds by the Committee of the Privy Council for Medical Research. A grant-in-aid of £148,000 was provided by Parliament for the Medical Research Council during the year. Of this sum £10,000 was used for administration. The expenses of the National Institute for Medical Research at Hampstead and of the farm laboratories associated with it, the salaries of the scientific staff and the expenses of research work done by them or by workers temporarily attached amounted to £52,000. For research in different centres, statistical inquiries and the investigations of the Industrial Research Board £86,000 was provided. Private benefactors and various public bodies interested in research augmented the funds of the Council to the extent of £20,000. Most of the grants made by the Council have been to approved workers in universities and research centres and but a small part has been used by men engaged in clinical work. This has a bearing on the conclusions drawn by the Council in regard to experimental medicine.

The question discussed by the Council is whether there is a science of experimental medicine in which the actual material for study is the human subject, or whether scientific work by the physician or the surgeon is limited to the application in his art of scientific results worked out elsewhere in the laboratory and delivered to him for use. It must be pointed out that a distinction is made between clinical research and laboratory research in which observation of human beings plays no part. The Council draws a clear distinction between the work of a physician engaged in practice and the work of a man "engaged in advancing knowledge." The choice of words is a little unhappy, for there is no doubt that the practising physician can by the accuracy of his clinical observation advance knowledge. It is obvious, however, what is meant. In the opinion of the Council the proper performance of either of these two kinds of work tends to exclude the proper performance of the other. "The training for one is compatible with the training of the other only along part of the way and beyond that it is incompatible with it." We would go further and would state that the training for each, at any rate along part of the way, is essential for the successful accomplishment of the other. According to the Council, the consultant physician, and much more the general practitioner, is devoted essentially and primarily to the task of diagnosis—the diagnosis of the condition of a particular patient; thereafter he is concerned with the treatment of a particular individual; his skill is rooted in scientific knowledge and he will make use of the latest results of science, but he cannot reasonably expect to be able to make the advancement of scientific knowledge a primary object of his practice. The research worker in the clinical field, it is further stated, must ignore individuality, obliterate it by multiplying cases and study as such the disease process which is in question; he must, of course, since human beings are the material of his study, learn "to deal with them with the same unselfish solicitude as that of any of his brother physicians"; his thoughts must be concentrated

chiefly on the task of getting new knowledge by every experimental method open to him. There is no training ground which will make the clinical research worker more fit for his task than that of general practice. Similarly there is nothing which will give to the practising physician independence of thought and make him refuse to accept unchallenged the written word of others more than a period of study in a laboratory, either of a clinical or general scientific character. The Council admits that certain exceptional individuals have been able to combine both careers, but is concerned in its statement with average persons and with the main features of the problem in its most normal form. It refers with legitimate pride to the work carried out by Sir Thomas Lewis and his associates in the first hospital "unit" and answers its own question with the statement that there is a science of experimental medicine.

This discussion by the Medical Research Council on experimental medicine is of considerable practical importance. If there is such a thing as a science of experimental medicine, and the views of the Medical Research Council in this regard may undoubtedly be accepted, the next question is the recruiting of those who are suited for the work, and the provision of a career. So far the response in Great Britain has not been good. This is ascribed on the one hand to the novelty of the position and on the other to the financial attractions of medical practice or the brighter prospects associated with laboratory work and an academical career in the university world. It is the intention of the Council to recruit workers suited to a career in clinical research, to help them in a period of training and to increase the number of workers in the clinical field upon their permanent staff. The Council has the funds to enable it to do this; some of the moneys doled out to isolated workers, as previously mentioned, could be put to better use in the payment of adequately trained permanent workers.

Lessons may be learned in Australia from the work of the Medical Research Council in England. The Council has referred with pride to the work of Lewis and his associates. It has also pointed out that, while the appointment of university "units" has been an unqualified success from the teaching point of view, the absorption of the university "units" in the work of general teaching has tended to prevent the provision of the essential conditions for research work within the unit. The new full-time appointments of professors of medicine and surgery within the University of Sydney offer possibilities similar to those of the professorships in university "units" in England. It is to be hoped that routine teaching in Sydney will not be allowed to overshadow the provision of facilities for research and its prosecution.

Current Comment.

PROPHYLAXIS OF CARCINOMA VENTRICULI.

DESPITE the aids to diagnosis provided by vastly improved radiological and biochemical methods, despite advances in surgical technique and the realization of the necessity for early operation, despite improved education of the general public and their inherent fear of malignant disease, despite much investigation and more speculation, cancer of the stomach, with so many and powerful forces of modern science arrayed against it, emerges victorious from most of its encounters and continues to take an increasing toll of human life.

In the absence of an understanding of ætiology, the possibility of scientific prophylaxis must be remote. The cause of cancer, as of many diseases, remains hidden in a mist of half-knowledge and its prevention is difficult and perhaps impracticable. Little but confusion has arisen out of the mass of argument for and against the numerous theories concerning circumstances and conditions at various times suggested as precursors or factors involved in the causation of cancer of the stomach.

It is interesting, therefore, to read the views of Arthur F. Hurst who in his Schorstein Lecture for 1929 stresses the need for a study of the precursors of *carcinoma ventriculi* and suggests methods of prophylaxis.[1] He believes that cancer never invades a healthy stomach and that its most frequent precursor is chronic gastritis which, rarely giving rise to striking symptoms, may be recognized by its associated achlorhydria and the presence of mucus. Carcinoma is not always accompanied by absence of free hydrochloric acid and Hurst knows of no instance in which the onset of the disease was followed by diminution in this acid. He believes that achlorhydria is a precursor and not a consequence of cancer, arguing that if his view were incorrect, the prognosis after operation when hydrochloric acid is undiminished should be more favour-

[1] *The Lancet*, November 16, 1929.

able than when hydrochloric acid is absent; that this is not so he endeavours to show by statistics.

He cites instances which go to prove that chronic gastric ulceration may acquire a malignant character and estimates that 20% of gastric cancers are preceded by ulcer. Simple adenoma and polyposis (probably a result of hypertrophic gastritis), while playing their part in the causation of cancer, are of less importance.

From a study of the situations in which cancer of the stomach most commonly occurs, it is obvious that some factor other than those already cited must be concerned. Hurst expresses the opinion that this factor is probably friction. He points out that there is little friction in the body of the stomach, but that it increases in intensity distally until at the pylorus the peristaltic waves produce definite churning movements.

Prophylaxis according to Hurst is possible and depends on a recognition of the precancerous conditions. The chief exciting causes of chronic gastritis and chronic ulcer—the two most important precursors of cancer—are identical and may be readily dealt with, hence prevention of these two conditions should present little difficulty. Attention to oral hygiene, removal of septic tonsils, treatment of sinusitis, regular meals, thorough mastication and avoidance of excess of food roughage are among the measures advocated by Hurst. Treatment of gastric ulcer and chronic gastritis immediately they are recognized is, of course, an obvious necessity.

Malignant degeneration of gastric ulcer has been a much debated question. Some investigators, notably at the Mayo Clinic, found evidence of malignant disease in as many as 75% of ulcers examined. Such high figures were probably due to the misleading appearances of certain epithelial inclusions which are apt to become involved in the fibrous tissue of ulcers and to simulate cancer. While admitting that malignant changes may develop in an ulcer, Maclean in England and Devine in Australia, on the other hand, believe that this development is extremely rare.

The association of *carcinoma ventriculi* with achlorhydria has long been recognized, but never understood. Maclean is of the opinion that the achlorhydria is due to cancer. Hurst is equally strongly convinced that the achlorhydria is *ante hoc* rather than *post hoc*.

Hurst does not state which tests for hydrochloric acid were used in his investigations and the possibility of confusion with lactic acid must not be overlooked. It is notable that he does not mention lactic acid at all in his lecture.

Achlorhydria, associated, as it is apt to be, with *carcinoma ventriculi*, pernicious anæmia, subacute combined degeneration of the cord or Hunterian glossitis, is a fascinating subject and whether Hurst's contentions are right or wrong, his suggested methods of prophylaxis should be useful in the prevention of disease of the stomach generally and are certainly worthy of a practical test. Should this Schorstein Lecture but stimulate further investigation into the causation of a dread disease it will have done a valuable service.

ABSORPTION OF GLUCOSE FROM THE RECTUM.

ALTHOUGH the rectal administration of glucose solutions has been widely adopted, it has, according to J. J. Pressman,[1] had little control study. He has investigated the absorption of glucose from the rectum. He gave to seven fasting patients eighty grammes of glucose by the rectum and on the following day an equal quantity by the mouth. He then determined the blood sugar content. He found that when glucose was given by mouth, there was an average maximum rise of sixty-five milligrammes per hundred cubic centimetres of blood. After rectal administration, on the other hand, no such rise occurred, but the average blood sugar level fell after four hours from 106 to 92 milligrammes. Under otherwise similar conditions, when saline solution was substituted, there was a drop of only four milligrammes. These findings are interpreted by the suggestion that sufficient absorption had taken place to stimulate insulin production, that an increased metabolism of previously stored sugar occurred, but that the absorption from the rectum had not been sufficiently rapid to replace the body sugars so destroyed. This explanation is probably the correct one. Pressman states that a rise in the respiratory quotient and the lowering of the blood sugar are evidence of an increase in sugar metabolism and call for the explanation that he offers. Jordan has shown that hyperinsulinism occurs when small quantities are introduced into the body. Pressman determined the sugar content of the stools of several patients and found that on an average 24% of the glucose was present in the stool at the end of four hours. In order to determine how much fermentation might be expected to take place in the rectum, he combined glucose and fæces and incubated them for thirteen hours. When specimens were examined after a lapse of seven hours, it was found that destruction of nearly 90% of the glucose had taken place. Pressman states that by a comparison of his figures with those in the literature it is obvious that the concentration and period of time devoted to the instillation are not factors in the ultimate result and that it cannot be argued that absorption does take place, but takes place too slowly to be reflected in an increase of the sugar content of the blood. He points out that repeated feedings by mouth of small quantities of glucose result in an increase of the blood sugar. It is obvious that absorption of glucose, albeit in small amounts, does take place from the rectum. Other observers have found an increase in the blood sugar and not a decrease. The insulin formation will influence the blood sugar content. This has to be thought of when therapy by glucose is considered.

[1] *The American Journal of the Medical Sciences*, April, 1930.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Inflammatory Reactions in the Liver.

GEORGE M. HIGGINS AND GEORGE T. MURPHY (*Archives of Pathology*, March, 1930) have studied experimentally induced localized inflammatory reactions in the liver of albino rats. They have given intravenous injections of a suspension of graphite. The quantities given varied from two or three to twenty-five cubic centimetres per kilogram of body weight. They conclude that the Kupffer cell is not an ordinary endothelial cell, but rather a connective tissue cell of the histiocytic series. They doubt whether typical capillary endothelium exists in hepatic sinusoids. After phagocytosis of particles of graphite the ingested material is agglomerated into larger granules distributed within the cytoplasm of the phagocytic cell. Within an hour of the production of an aseptic lesion in the liver, the Kupffer cells retract their processes, become swollen and spherical and often detach themselves and pass into the blood stream as free mononuclear cells. These macrophages become free wandering histiocytes. Local histiocytes in the interlobular spaces rapidly proliferate and form many free wandering cells or polyblasts. Typical monocytes accumulate in the perivascular spaces and may arise as a transformation of migrated lymphocytes. Polymorphonuclear leucocytes appear within the necrotic lesion and are abundant after twenty-four hours; they are restricted to the inner regions of the lesion and do not change into other cells. Mononuclear infiltration forms a capsule to the lesion and this capsule is transformed into a prominent heavily carbonized wall. Mononuclear cells arise from two sources, from the agranulocytes of the blood stream and from the local histiocytes of the periportal tissue, including detached Kupffer cells. Fibroblasts arise from mononuclear cells which form the wall of the lesion. Fibroblasts containing graphite are transformed macrophages, developed largely from Kupffer cells. Fibroblasts containing no graphite develop from the histiocytes of the periportal spaces and the transformed lymphocytes and monocytes of the blood stream.

Changes in the Spleen in Peritonitis.

P. GOHRBANDT (*Virchow's Archiv für Pathologische Anatomie und Physiologie und für Klinische Medizin*, June, 1929) has carried out experimental observations into the changes in the spleen produced by peritonitis. His experiments were carried out on dogs and he divided these into four groups according to the method used for the production of peritonitis. The methods were: wounding of the stomach, opening the colon, the production of an intraperitoneal biliary fistula, the introduction in pure culture of fungi pathogenic to the dog. On macroscopical examination no recognizable changes were found. On microscopical examination, on the other hand, changes were extensive. The amount of blood in the tissues was variable. Pronounced follicular necrosis was present as well as definite nuclear and cellular destruction in the lymph nodules and to a less extent in the cells of the pulp. An increased deposit of lipoid was noted particularly in the reticulum cells of the pulp and in the lymph nodules. Deposits of lipoid were also found in the cells of the blood sinus and perivascular lymph vessels. No lipoid was found surrounding the capillaries. There was a considerable increase of hæmosiderin in the reticulum cells and in the endothelium. A leucocytic reaction occurred in the first few hours of the infection. Subsequently there was a decrease and rearrangement of the oxydase cells and eventually there appeared a typical arrangement—a broad perifolilicular zone and subcapsular aggregation with considerable involvement of the capsule and with almost complete disappearance of the oxydase cells from the pulp. The disappearance of the cells is to be explained, not by a smaller addition, but by an increased removal of cells. Their perifollicular localization is to be regarded as the reaction to the toxin which attacks the lymph nodules particularly. As a rule there was an enormous increase of the true as well as lymphoblastic plasma cells. These are not to be looked upon as a particular kind of cell, since every fixed spleen cell can become a plasma cell under certain functional demands. In two animals a myeloid metaplasia was found. The arrangement of these cells points to their origin from undifferentiated adventitial cells.

Tissue Reaction in Rheumatic Disorders.

VINCENT COATES (*Proceedings of the Royal Society of Medicine*, March, 1930) believes that orthodox rheumatic infection and multiple infective arthritis of unknown origin are but varied expressions of the same disease and that circumstance, organ inferiority and immunity decide the site of election and the exuberance of the reaction. He allies orthodox and heterodox rheumatic infection for two main reasons: First, because many disorders of an infective nature are prone to exhibit widely divergent manifestations and, secondly, because one condition merges into another and there occur common symptoms, complications and cardinal signs. The author holds that the tissue reaction is specific, not only for orthodox, but also for heterodox rheumatic infection. He describes the stages by which the conclusion has been reached that orthodox rheumatic infection gives rise to a specific tissue reaction. He also quotes from the work of Hawthorne and of Coombs and himself to show that heterodox rheumatic infection gives a similar reaction to that of orthodox infection. He describes and illustrates sections of a bursa removed from a patient with infective arthritis. The bursa manifested subacute productive inflammation, perivascular in distribution, and containing localized inflammatory nodules rich in young capillaries and large endothelial or histiocytic cells. These nodules did not differ essentially in structure from those of acute rheumatism.

Hyperthyreoidism and Inanition.

J. P. SIMONDS AND W. W. BRANDES (*Archives of Pathology*, February, 1930) have made an investigation of the effect of experimental hyperthyreoidism and of inanition on the heart, liver and kidneys. They studied the body weights and the heart, liver and kidney weights of thirty-one normal adult dogs and nine adult dogs, each of which was given ten grammes of desiccated thyreoid every day and one dog which received twenty grammes a day for periods ranging from thirty-two to one hundred days. All the animals experimented upon were kept on the same diet and under the same conditions as the normal controls. For comparison corresponding weights were collected from studies on inanition. The authors concluded that in both inanition and experimental hyperthyreoidism the animal as a whole loses weight. The part played by the individual organs, however, is different in the two conditions. In starvation the heart loses weight in about the same or somewhat smaller proportion than that of the body as a whole. In experimental hyperthyreoidism, not only may the heart not lose weight, but in some animals it may appear to increase in size, for the actual weight after prolonged thyreoid feeding may be greater than the estimated heart weight at the beginning of the experiment. The effects of inanition and of experimental hyperthyreoidism on the kidneys corresponds rather closely to their respective effects on the heart. In inanition the liver loses weight in greater proportion than the body as a whole; this loss is closely related to the disappearance of glycogen from that organ. In experimental hyperthyreoidism the liver practically maintains its original weight in spite of its loss of glycogen. The relative, if not absolute, hypertrophy of the heart, liver and kidneys in experimental hyperthyreoidism is probably due to the combined effect of increased function and augmented blood flow. Increased function and augmented blood flow are essential factors in hypertrophy.

Generalized Osteitis Fibrosa with Parathyreoid Hyperplasia.

A. M. DRENNAN (*The Journal of Pathology and Bacteriology*, January, 1930) records a case of *osteitis fibrosa* with cystic changes in the bones and a large mass of parathyreoid tissue. The bony changes were similar to those described by other authors. There was nothing in the history to suggest a cause to the condition. The thyreoid changes were degenerative

and unlikely *per se* to have any relation to the bone disease. The mass of parathyreoid tissue (present in the position of the right lower parathyreoid) seemed to be either tumour growth or hyperplasia of the pre-existing gland to account for absence or loss of the other parathyreoids. The author points out that the relationship of the parathyreoids to calcium metabolism is still obscure and, as no record was made in his case of calcium intake or output and no chemical examination of the blood or organs was attempted, he contents himself with recording the morphological findings.

MORPHOLOGY.

The Mesenteric Circulation.

A. J. COKKINIS (*Journal of Anatomy*, Volume LXIV, Part II, 1930) makes observations on the mesenteric circulation. He finds first of all that the *vasa recta* in the small intestine divides into two branches which, instead of passing from opposite sides of the mesenteric border to encircle the gut, as described by French anatomists, pass always on the same side of the gut. Usually the two branches of the *vasa recta* passing on the one side are followed by two branches passing to the other and there is an alternation; but this is by no means constant. The antimesenteric border of the gut is always found free from visible vessels. As regards the venous drainage of the intestine, the jejunal and ileal veins were found to be more variable in number than the arteries and many variations were found in the manner of termination of some of the main venous tributaries; especially in the ileo-caecal region was disagreement found with accepted teaching. It is usually stated that the ileo-caecal region is drained by an "ileo-colic vein" which is said to accompany the ileo-colic artery and open into the superior mesenteric vein some distance up the root of the mesentery. The author found, however, that in the majority of bodies examined the venous radicles of the ileo-caecal region formed the commencement of the superior mesenteric vein itself, while the small vein accompanying the ileo-colic artery was merely an anastomotic communication between the superior mesenteric vein and the marginal arcade along the ascending colon. As regards the blood supply of the great omentum, this was found to be from the gastro-epiploic vessels and very little, if any, came from the middle colic vessels, except where pathological adhesions were present. As regards the collateral circulation of the mesentery, it was found that ligature of jejunal and ileal rami, singly or in contiguous groups of two and even three, in no instance prevented injection material from reaching the entire territory of the occluded vessels by collateral channels. Ligature, however, of a terminal arcade completely obstructed the flow of ink to the guts supplied by them and there was a short margin between the non-injected gut and mesentery and the injected gut; similarly with the *vasa recta*. In conclusion the author states that: (i) The collateral circulation within the mesentery itself, up to and including the terminal row of arcades, is very free and occlusion of quite large vessels fails to prevent injections at fractional pressures reaching every part of their territory. (ii) The collateral circulation stops with the terminal row of arcades. Beyond this there is absolutely no anastomosis, either between the *vasa recta* in the mesentery or between the ramifying vessels on the gut wall. (iii) The external anastomoses of the mesenteric vessels are as inefficient as their anatomical construction would lead us to expect.

Histological Changes in the Epithelium of the Human Vagina and the Menstrual Cycle.

HISTOLOGICAL changes in the epithelium of the human vagina correlated with the menstrual cycle are described by B. G. Smith (*Anatomical Record*, September, 1929). Material was obtained *post mortem* from a *nullipara*, aged twenty-three years (Case I) and a further series from a *nullipara*, aged thirty years (Case II), was used for comparison. In the absence of menstrual history of the former, examination of the uterus showed a very early premenstrual condition which was confirmed by the presence in the ovary of a very recent *corpus luteum*. The vaginal epithelium varies from typical stratified squamous to an epithelium differentiated into three zones, the middle one of which consists of cells undergoing cornification. In the latter case sections of the vagina alone are examined, but it fits readily into Dierk's series as a very late premenstrual stage; the epithelium closely resembles that of Case I, differing only in that the outer layers are becoming desquamated and that a leucocytic infiltration is taking place. In both instances the outstanding feature of the epithelium is its great variation in different parts of the vaginal wall. A comparison is made with the changes of the vaginal epithelium associated with the œstrous cycle in lower mammals. A brief description is given of the epithelium of the uterine tubes of Case I, in which is seen extrusion of entire epithelial cells into the lumen of the tube, suggesting the possibility of cyclic changes in this region also, of a similar nature to those described in the mouse.

The Form and Position of the Empty Stomach.

ROBERT ORTON MOODY, ROSCOE G. VAN NUYS AND CAROL H. KIDDER (*Anatomical Record*, Volume XLIV, Number 4, 1929) have examined the form and position of the empty stomach in several hundred healthy young adult students in the University of California. The stomach was not quite empty, one mouth-full of a barium-sulphate-malted-milk mixture being given about five minutes before exposure to facilitate radiography. The authors remarked also that none of the subjects examined had a history of chronic intestinal trouble or constipation; they confirmed the generally accepted belief that in the living the form and position of the empty stomach vary greatly in different individuals and change in an individual with change in posture. The form of the empty stomach fell into four types: Reverse L, cylindrical, J-shaped and roughly crescentic. The lowest part of the greater curvature of the empty stomach may normally occur as high as the upper half of the twelfth thoracic vertebral body or as low as the upper half of the first sacral vertebral body; it may be found below the interiliac line with the subject erect, prone or supine. The pylorus of the empty stomach in the living has a wide range of occurrence; it may be found as high as the upper part of the twelfth thoracic vertebra or as low as the lower half of the fifth lumbar vertebræ; it may lie above the transpyloric plane, but is most often below it; it may lie on the mid-line or on the right or left of the vertebral column. The greater curvature and the pylorus are usually lower in the female than in the male. A number of observations were also made as to the relations of the first part of the duodenum to the head of the pancreas in the cadaver and some of the varying conditions found are figured in a plate.

Localizing Inorganic Substances in Microscopical Sections: The Microincineration Method.

A. POLICARD AND H. OKKELS (*Anatomical Record*, Volume XLIV, Number 4, 1930) have described a technique of making incinerated microscopical sections. By this method ordinary histochemical reactions can be controlled and counterverified; also ash preparations can be employed to advantage when the total content of mineral matter in any given tissue is to be studied. Concise determination of the distinct inorganic compounds may also be estimated. These workers further describe in detail the advantages and disadvantages of their new methods and conclude that microincineration will be a helpful instrument of advance, not only to histochemistry, but also to many other branches of biological research. The article is well illustrated with a series of microphotographs.

Observations on Injured Cells.

E. KUSTER (*Protoplasma*, Volume VII, 1929, page 150) concludes that plasma membranes of an injured cell and vacuoles are able to retain their semipermeability for some time after stimulation has occurred. It was found that the membrane of a vacuole which has been cut open, is capable of contracting, so that a completely closed vacuole is formed and the part cut off containing an open vacuole soon dies. When dealing with only slightly injured cells, it was discovered that the whole cell, as well as the vacuole membrane, may be made to show osmotic contraction.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Darling Building, University of Adelaide, on March 27, 1930, DR. H. GILBERT, the President, in the chair.

A Disease Resembling Laryngeal Diphtheria.

DR. FRANK BEARE read a paper entitled: "A Series of Cases Resembling Laryngeal Diphtheria" (see page 668).

DR. H. M. JAY said that he had been very interested in Dr. Beare's report, but he could add nothing in the way of confirmation to what had been said, as he had not come across any case of this type. It seemed to him from Dr. Beare's description that the condition had some resemblance to what was being described in American journals as agranulocytic angina, except that the larynx instead of the pharynx was being attacked. He suggested that it would be interesting to make a differential blood count in these cases in order that the number of granular leucocytes (characteristically diminished in agranulocytic angina) might be determined and any relationship between the two affections established.

DR. A. R. SOUTHWOOD referred to the occurrence of cases of ulcer-membranous laryngitis during the influenza epidemic of 1918. Tracheotomy was practically useless, for the inflammation was not usually restricted to the larynx, but involved the trachea and bronchi. Often also there was associated bronchopneumonia. *Post mortem* examinations in such cases revealed an acute inflammatory œdema of the respiratory tract, with a rather characteristic plum-coloured engorgement. Death was due to general toxæmia and acute cardiac failure rather than to respiratory obstruction.

DR. PERCIVAL CHERRY stated that in about 1912 he had sent a child from Tailem Bend to the Adelaide Children's Hospital with what was clinically a condition of laryngeal diphtheria. The child had been brought down from the Murray lands by train. He had examined it in the railway carriage at Tailem Bend, isolated it therein and sent it on to Adelaide. He had advised the railway authorities to have the compartment disinfected. Dr. L. W. Jeffries had been house surgeon at the time at the Children's Hospital and on telephoning him a day or two afterwards Dr. Cherry had been surprised to learn that bacteriologically the condition had proved not to be diphtheritic. Dr. Jeffries had informed him that there had been quite a number of patients with a similar condition admitted about that time. If Dr. Beare looked up the records of the Children's Hospital for that period, he might find that these infections were similar to those that he had reported that night.

Vaccine Therapy.

DR. D. L. BARLOW read a paper entitled: "Vaccine Therapy" (see page 671).

DR. GILBERT BROWN congratulated Dr. Barlow on his interesting and instructive paper. Probably all the members of the Branch had worked miracles with vaccines at times, the gonorrhœal joint cleared up in twenty-four hours, whooping cough was cured in a week, arthritis was relieved quickly by a vaccine taken from a tonsil and the winter colds were banished by prophylactic vaccination. Unfortunately these dramatic results did not "come off" with sufficient regularity and vaccine treatment was often disappointing. Consequently medical men were divided into three camps: the enthusiasts who cured everything with vaccines, the pessimists who believed that vaccines never did any good, and the wobblers who used vaccines with an open mind and expected occasional cures. If Dr. Barlow could help to clear up the mystery and enable the miracles to be worked more frequently, and his paper gave some hope of this, he would earn the gratitude of all present.

Dr. Brown said that he would be glad if some explanation could be given of the varying results that he had obtained in treating whooping cough with "Pertussis Immunogen Combined." Two years ago he had had uniformly good results and all patients were cured by from five to seven injections; but the following year the benefit received from the injections had been very slight. He was assured by the makers that the product was unchanged and the amounts used were the same. Was the difference in results due to a variation in the strain of the organism and that this particular strain was absent from the material from which the immunogen was prepared?

DR. H. M. JAY said that he would like to substantiate what Dr. Barlow had told the meeting regarding sensitiveness to foreign proteins. In nasal work he came across a considerable number of patients suffering from "hay fever" and allied troubles. His patients had all been tested and treated by Dr. Barlow and he had yet to hear of one who was not satisfied with the results obtained. Personally he had the greatest faith in this work, provided that it was properly carried out.

DR. F. LE MESSURIER asked how cultures of whooping cough bacilli could be obtained. He was sure from his experience that the best results in treatment were secured by using freshly prepared vaccines.

DR. J. CLOSE said he had often wondered about the specificity of typhoid vaccine when used during the height of typhoid fever. He had not treated a patient with typhoid fever for some years, but during his practice at Port Pirie he had come in contact with a lot of patients with this disease, as the sanitation there was at that time very primitive. He and his colleagues had been very thrilled at the dramatic result which frequently followed an intravenous injection or two of the vaccine, but he had often wondered whether *Bacillus coli communis* vaccine or one of the commercial proteins might not have given a similar result. He was unaware of the methods of treating these patients at the Adelaide Hospital, but no doubt Dr. Barlow could tell him whether non-specific proteins were in favour. One case of his had made a great impression. It had been preceded by a disastrous case in which a leak had been suspected, but after consultation with a colleague it had been decided to wait a little while to make sure; the wait had unfortunately been too long and at laparotomy an hour after the abdomen had been found flooded with intestinal contents and the patient had not recovered. When the patient in question developed similar signs of perforation, distended, rigid and tender abdomen, vomiting and very rapid pulse, it had been decided after consultation with several colleagues to operate at once. At operation nothing in the way of complications had been found and the abdomen had been sewn up again. The result had been better than that seen with vaccines; the temperature, rapid pulse, distended abdomen and all the other alarming signs had disappeared at once and the patient had made an astoundingly rapid recovery. It seemed possible that the liberation and absorption of some non-specific body, such as thrombo-kinase, had worked the oracle. Strangely enough, immediately after this case he had read a report in THE MEDICAL JOURNAL OF AUSTRALIA, by Dr. Ray, of an exactly similar case at the Adelaide Hospital.

DR. F. BEARE said that failure to grow whooping cough bacilli had been a common experience at the hospital departments and asked what method would succeed.

DR. H. K. FRY stated that he considered that pessimism in regard to the use of vaccines was justifiable in the present state of knowledge and practice. In the first place there was no scientific method of checking the response to the vaccine. The original method of calculating the opsonic index had gone by the board. The estimation of immune body formation was no criterion, for in one of the most successful applications of the treatment, the use of a staphylococcal vaccine for boils, no immune body was formed. Secondly, from the discussion of the chemical nature of bacterial antigens in Jordan and Falk's symposium, "The Newer Knowledge of Bacteriology and Immunology," he had understood that the specific antigens usually were destroyed in the processes of preparing the vaccines. In a number of cases, for example, the specific antigen had been found to be a labile protein-carbohydrate substance. The carbohydrate radicle supplied the specific

factor, but, separated from the protein, was not an antigen itself. The protein radicle in the absence of the carbohydrate combination formed an antigen with little specific value. The protein-carbohydrate linking was broken down by the ordinary methods of sterilizing a bacterial emulsion.

DR. H. GILBERT recalled that he had worked with Sir Almroth Wright in the early days of vaccine therapy and on his return to Adelaide had prepared and used vaccines for such conditions as boils, carbuncles and pustular rashes with great benefit. He wondered whether the method of sterilizing vaccines with chloroform had impaired the usefulness of many vaccines.

In reply Dr. Barlow stated that there was usually much uncertainty as to the likelihood of obtaining results from the use of stock vaccines. No suggestion had been made that results could be obtained in every case and gonococcal arthritis had not been mentioned as an indication for vaccine therapy.

In reply to Dr. Beare and Dr. Le Messurier he said that in regard to the cultivation of the whooping cough bacilli special care was required in the preparation of the media and also in keeping cultures at incubator temperature from the moment of inoculation.

In reply to Dr. Close he said that organisms other than typhoid bacilli produced similar effects in the treatment of typhoid fever by vaccines given intravenously. Non-bacterial proteins were also capable of doing this. It was possible that in the case mentioned the operation had a similar effect.

In reply to Dr. Fry he said that he did not agree that the bacteria underwent chemical disintegration during the ordinary process of sterilization, as suggested. In reply to the theoretical argument that vaccines could not be expected to do good, he quoted cases in which vaccine therapy had been conspicuously successful after other treatment had failed.

Referring to Dr. Gilbert's remarks on the apparently better results obtained when using Almroth Wright's original method of sterilizing by means of heat, instead of by the use of chloroform as was sometimes done, he had always employed the heat method, taking care to avoid any excess.

Interstitial Cervical Fibroid.

DR. BRIAN SWIFT showed a specimen of a uterus with a large interstitial cervical fibroid which had been removed from a woman, aged forty-five. Her only complaint had been symptomless uterine hæmorrhage during the last three months and for her anæmia she had had one blood transfusion a week before operation. On abdominal examination a hard, irregular shaped tumour had been felt, about the size of a cricket ball, arising from the pelvis. On vaginal examination a large, hard mass had been encountered which filled up the pelvis and was firmly fixed. The cervix had been found to be thinned out into a "half moon" shape and was up behind the pubic bone. There had been no cervical canal. These findings had been typical of a cervical fibroid arising in the posterior lip of the cervix.

At operation the abdominal tumour had been found to be the body of the uterus studded with numerous fibroids and it had been sitting on top of the large cervical fibroid which was tightly jammed in the pelvis and could not be moved. The tubes, round ligaments and broad ligaments on each side had been divided, but still the tumour could not be moved. A transverse incision had then been made in the posterior uterine wall low down and the capsule of the fibroid had been found. The fibroid had been enucleated with ease and with very little hæmorrhage. A total hysterectomy had then been done quite easily, but the posterior portion of the fibroid capsule had been left as it was firmly adherent to the rectum nearly as far as the anal canal. A gauze drain had been left in the vagina. The woman had made an uninterrupted recovery. The specimen demonstrated the thinned out cervix and the large anterior wall of the capsule which was continuous with the external *os* which was a thin lip of tissue. The fibroid was large and measured 16·25 by 13·75 centimetres (six and a half by five and a half inches) and was globular.

Dr. Swift said that the reason why the patient had had no urinary or bowel symptoms was that the fibroid had most probably grown slowly, otherwise it was difficult to understand how a tumour of such a size could cause no pressure symptoms.

Radium in Menopausal Hæmorrhage.

Dr. Brian Swift's second specimen was a uterus which had been removed from a woman, aged thirty-nine, for continuous uterine hæmorrhage. Her uterus had been curetted for menorrhagia of ten years' duration in January, 1930, and twenty milligrammes of radium had been introduced into the uterus and left for forty-eight hours. The radium had been in the form of two ten milligramme needles screened with 0·5 milligramme of platinum and placed in a rubber tube one millimetre thick in tandem fashion and tied in position. Since January continuous bleeding had occurred and for this reason it had been decided to remove the uterus. A total hysterectomy had been done and the specimen demonstrated two interesting points. First, the presence of a small submucous fibroid situated at the left cornua where it was impossible to be felt by means of a curette and it had therefore been missed at the original curetting. If it had been felt, then radium would not have been introduced, as a submucous fibroid would not respond to radium treatment. Secondly, a small white area was seen in the endometrium just internal to the internal *os*. This area was hard and on section proved to be an area of necrosis which not only involved the endometrium but also extended to the muscle. It was most probably a radium burn.

Dr. Swift said that burns of this description might possibly be the cause of the usual excessive first menstrual loss after the insertion of radium for uterine hæmorrhage, the separation of a slough from such an area being the cause. Dr. Swift also mentioned that it was difficult to tell how close the radium was to the endometrium at various positions in the uterine cavity and so how close to the ovaries. In large cavities the dose could be bigger, whilst in areas near the cervical canal the radium would be in intimate contact with the endometrium and so the dose would have to be smaller to avoid burns. Dr. Swift said, however, that the endometrium of this specimen was quite normal except at this one spot. This helped to prove that the radium acted on the ovaries and provoked an artificial menopause and stopped the uterine hæmorrhage for this reason rather than for its action "supposed" on the endometrium.

Skiagrams.

DR. L. O. BETTS showed two X ray films. The first was that of a spine of a man, aged sixty-nine, who four years previously had strained his back lifting a gate post. At the time something had been heard to crack in his back and he had had a painful back which necessitated recumbency for a few days. Since then he had had some stiffness and mild discomfort in the lumbar region. The skiagram revealed an old crush fracture of the twelfth dorsal vertebra. There was no history of any other injury that might have caused the fracture and the film was shown to illustrate the fact that these fractures were more common than was usually recognized and that they passed undiagnosed.

The second films showed the process of repair in a birth fracture of the femur over a period of six months. Although there had been 2·5 centimetres (an inch) of shortening in the original fracture with considerable misalignment and deformity, at six months the alignment had been almost perfect and all the shortening had been made up.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on April 4, 1930, DR. S. F. MCDONALD, the President, in the chair.

Albuminuria and Eclampsia.

DR. F. HOPE MICHÔD read a paper entitled: "Albuminuria and Eclampsia" (see page 679).

DR. A. H. MARKS congratulated Dr. Michôd on his valuable paper. He considered the subject to be one of great importance. Dr. Marks said that he would like to

see a comparison made of the incidence of eclampsia in different parts of the world, for example, India with its hot climate and vegetable diet.

Dr. Marks agreed that the uterus should be emptied if the kidneys were damaged; the difficulty was that these patients generally became pregnant again. There were sociological difficulties in ligaturing the tubes, but he considered it justifiable in a woman who already had a family. He hoped Dr. Michôd would continue his researches.

DR. KENNETH WILSON quoted the figures of the Rotunda Hospital for 1928. There had been 2,256 admissions. There had been 360 patients (or 15·9%) with albuminuria and one maternal death, a rate of 0·27%. The fœtal death rate had been 1·9%. Twenty-one patients or 0·9% had suffered from preeclampsia (or eclampsism). This was described as a condition in which all the clinical features of eclampsia were present in the absence of fits. The maternal death rate had been nil and the fœtal death rate 23·8%. Twelve patients had suffered from eclampsia, a rate of 0·5%. The maternal death rate had been 16·6% and the fœtal death rate 33·3%.

There was no division of albuminuria into nephritic and toxæmic types, as it was held that the prognosis and treatment were much the same in both groups.

With regard to the prognosis, it was held that the patient should be advised against pregnancy until the urine was free from albumin and that once a patient had had albuminuria she was liable to a recurrence.

Dr. Wilson said in reference to Dr. Paramore, of Rugby, whom Dr. Michôd had quoted, that he had had the pleasure of listening to him at the Dublin Congress of Obstetrics and Gynæcology last year and wished to draw the attention of members to the fact that Dr. Paramore had reported two patients with eclampsia treated solely by means of spinal anæsthesia with "Novocain"; both patients had recovered. This treatment was the logical sequence of his mechanistic theory and although two cases were not sufficient from which to draw conclusions, yet the theory was an interesting one.

Dr. Wilson wished to thank Dr. Michôd for his interesting paper and to record his appreciation of the amount of time and effort such a paper required.

DR. ELLIS MURPHY congratulated Dr. Michôd. He thought that in the future, when tests of renal function would become more sensitive, it would probably be found that albuminuria of pregnancy and eclampsia occurred only in those patients with some preceding renal disease.

He thought that before drastic measures were taken in the surgical prevention of pregnancy, one should be quite sure that very definite renal disease was present, in other words, chronic nephritis. Several tests helped in regard to the prognosis in such circumstances. A high blood urea could be inferred if the specific gravity range was less than three and a specific gravity that remained at about 1010, appeared to be of grave omen. Also if the maximum urea concentration in the specimens of urine were less than 2% and the minimum were less than 0·2% or 0·3% below this, the blood urea was almost sure to be raised.

In regard to the blood pressure, more reliance should be placed on the diastolic pressure than the systolic and a high pulse pressure was not of such grave significance as a smaller one with a high diastolic pressure which tended to remain very constant.

DR. H. S. WATERS congratulated Dr. Michôd on his paper with its mass of statistical detail. Dr. Waters was interested in *primiparæ* with albuminuria. He called attention to the contrast in cases number II and number X. Both of these *primiparæ* had advanced to the thirty-fifth week when in the one case surgical induction had been tried and in the other premature labour had resulted, but the result had been death to the fœtus. This practically sealed the doom for life for the couple having any family. He asked whether one should not avoid interference if at all possible, in the hope of getting a living child.

He thought that patients with eclampsia developing quickly in which fits occurred without previous albuminuria, seemed to do better than those whose eclampsia got worse gradually after prolonged treatment for albuminuria. He asked what was considered the best thing to do, with the object of getting a live child, with a *primipara* coming into her last months with albuminuria. Was there any indication in the fœtal heart of the time for interference? Also what was the cause of a woman successfully carrying a child until the end of the ninth month and then suddenly producing a macerated fœtus?

DR. S. F. MCDONALD said that he did not see much midwifery, but he did see women with kidney conditions who came to know whether they were fit to have any more pregnancies. He had seen only one of these patients who gave normal results to kidney tests.

The important points in examination in these circumstances were the blood urea, blood pressure, urea concentration and retinal examination. If the urea concentration were lower than 2·5, it meant gross damage to the kidneys. Rosenthal's test also was done if the patient had frequency of micturition.

The one woman he had seen as giving normal results to all tests and who had had no previous illnesses, had been advised that if she were normal for two years, she could safely have more pregnancies.

Another *primipara*, aged twenty-three years, had given a history of being well except for an attack of infantile paralysis which had affected both feet. She had had a concentration of over 2·0, a systolic blood pressure of 160 millimetres of mercury and albumin in the urine. She had disregarded advice, had become pregnant, had had fits at six months and died. A detailed history of childhood was necessary in all cases. He considered that most eclamptics were nephritics whose condition often resulted from lead poisoning or some disease in youth. If these patients were to be kept alive and mentally comfortable, the prevention of further pregnancies was most important.

Dr. Michôd in reply agreed with Dr. Marks that the practice of inducing abortion repeatedly in the same woman was to be deprecated. A better method was to make the patient consent to a "sterilizing" operation to follow immediately after the second abortion. Dr. Michôd also stated that the staff of the Crown Street Women's Hospital, Sydney, favoured a vaginal operation in preference to an abdominal.

In reply to Dr. Wilson's remarks Dr. Michôd said he considered that the prognosis for both the mother and the child was much worse in the nephritic patient and the percentages were difficult to compare when the separation into "toxæmic" and "nephritic" cases was made.

In reply to Dr. Waters, the two cases number II and number X were very similar. In number II the condition was considered to be a definite nephritis which had probably been in existence before the patient became pregnant. The patient in case number X would probably soon be placed in the nephritic group; six months had not yet elapsed since the confinement.

The reason for the woman who developed eclampsia without a prolonged albuminuria doing better than a woman who had albumin during the greater part of her pregnancy, was in accordance with the supposition that the eclamptic fits were due to the effect of the toxæmia on the cerebral cortex, the toxæmia probably developing late in the pregnancy and possibly in a previously healthy kidney. The prolonged albuminuria in Dr. Waters's other patient suggested that the kidney was either already nephritic or that else a considerable portion of the kidney had been already destroyed by the toxæmia. Dr. Michôd did not consider that the fœtal heart was any guide as to when to terminate the pregnancy, the termination of the pregnancy being more for the benefit of the mother than the fœtus.

Dr. S. F. McDonald's observations were in accordance with those of Dr. Michôd, particularly the view that most eclamptics were really latent nephritics.

ANNUAL MEETING.

THE ANNUAL MEETING OF THE WESTERN AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Hospital for the Insane, Claremont, on March 23, 1930, DR. J. J. HOLLAND, the President, in the chair.

Financial Statements.

The Honorary Treasurer, Dr. A. Syme Johnson, presented the balance sheet and financial report for the twelve months. These were received and adopted.

President's Address and the Annual Report.

In accordance with custom the retiring President combined his address with the report of the Council.

Membership of Branch.

The membership of the Branch has increased from 234 to 244.

It is with very much regret that I have to record the deaths of seven members of the Branch, namely:

Dr. Morton, Victoria Park; Dr. John Cuthbert, Perth; Dr. William Trethowan, Perth; Dr. McLean, Perth; Dr. Saw, Perth; Dr. Glanville, Swanbourne; Dr. Lyon Johnston, Mt. Hawthorn; Dr. Bateman (non-member).

We much regret the loss of so many medical men, especially the tragedies of the loss of Lyon Johnston and Ian McLean in their youth. But two names stand out in the list as having been distinguished members of the Branch, both in their professional attainments and their conduct as great examples to us all, namely, William Trethowan and Athel Saw. We would wish that the profession was entirely made up of men of their types.

Resignation of Dr. H. T. Kelsall.

It is with much regret the Council received the resignation of Dr. Kelsall on account of ill-health. Dr. Kelsall being one of the foundation members of the Branch, he was elected an honorary member.

Meetings of the Branch.

The meetings of the Branch have been well maintained. There were ten general meetings with an average attendance of thirty-three members. These meetings included clinical evenings kindly arranged by Dr. Anderson of the Perth Hospital and Dr. Male of the Children's Hospital.

Interesting papers were read by Dr. R. Williams, Dr. J. G. Hislop, Dr. Beveridge, Dr. J. J. Holland, Dr. L. A. Hayward and Dr. Alfred Webster.

During the year there was an interesting address by Dame Janet Campbell.

There was also a successful joint meeting with the Odontological Society.

The annual dinner which was held in August was also thoroughly successful.

The Branch had the honour of entertaining Sir Ewen Maclean at a smoke social in October, 1929.

Ethical Committee.

One meeting of the Ethical Committee was held during the year.

Meetings of Council.

The Council held eight meetings, with an average attendance of six members.

Much work was done by both the general meetings and the Council and the following are the main items considered:

Housing Accommodation for Branch.

A committee was appointed on April 3, 1929, consisting of the President, the Honorary Secretary, Dr. K. Moss, Dr. A. Syme Johnson and Dr. Stewart.

I moved in this matter many years ago, but very little has been done owing to the difficulties of financing the scheme and the tendency to narrow the area of choice to entirely within the busy portion of the city.

If I had my choice, I would purchase in the northern part of the city, say in Stirling Street or Aberdeen Street, where a large amount of capital is not required and where ample space could be secured to allow for the future building. I hope my successor, Dr. Baldwin Gill, will not be content with his year's work until he secures a site for the Branch.

Constitution of the Federal Council.

This is submitted to you tonight for confirmation, with the recommendation of the Council. The Federal Committee at present exists with two delegates from each State of the Commonwealth known as the Federal Committee of Australia, but they have no constitution or power or authority to control money. It is for this purpose that the constitution has been created. As each State of the Commonwealth has two representatives on the Federal Committee, we are well represented. The matter has been gone into by the Council and also the Federal representatives (Dr. Paton and Dr. Hadley) and you are now recommended to pass it.

The Medical Journal of Australia.

The local representative, Dr. J. Hislop, having resigned, the Council have approved of the appointment of Dr. J. P. Ainslie at the instigation of the Editor.

We much regret the death of the late Editor, Dr. H. W. Armit, who had a most worrying time in the management of the journal.

Hospital Act.

Last year the *Hospital Act* was brought forward, but did not get through Parliament. A scheme has been adopted by the Perth Hospital where weekly payments secure the benefit of hospital treatment. It is one part of the scheme that this is only accepted from those who are unable to pay for medical services outside. This is a matter not easy to check and great care will have to be used to prevent this being abused. It must be borne in mind that the Government, through the Hospital Committee advocating this scheme, are doing so at the expense of the medical profession who as members of the honorary staff are giving their services free and anyone abusing this scheme and getting treatment at the hospital when they are able to pay for it, are receiving medical services as a charity.

Australasian Medical Congress (British Medical Association), 1932.

An invitation has been issued by this Branch to the Federal Committee for the holding of the next Congress in Perth. The Vice-Chancellor of the University has kindly written intimating they would be prepared to grant the use of the University buildings for the Congress, such buildings should be completed in time.

I understand the South Australian Branch has also issued an invitation for the Congress and we are now waiting the decision from the Federal Committee.

I know that some members fear the failure of a Congress in Perth. As one who has advocated holding Congress here I am sure that we have nothing to fear. At the recent Congress in Sydney we had so many promises of help by members from other States that I am sure that you will all be pleased after its occurrence. We have the men with ability and energy here to carry it through and I have the assurance of the present Premier, Mr. Collier, that whatever Government is in power it will help most materially in entertaining visitors and there is a big chance of direct financial help.

Workers' Compensation Act.

At the end of last year the underwriters who had submitted few accounts for revision, complained they were being penalized by the profession in the smaller accounts and suggested the abolition of the Committee.

Your Council contended that lay people, including insurance companies, were not qualified to judge professional accounts and further that they were prepared to consider any account in question no matter how small. Although the insurance companies had a schedule to guide them in passing accounts, they evidently took the Association at its word and for the last three months have sent every individual account to the Special Committee for revision. In the month of February these numbered about 250. Apart from the great increase in clerical work and cost of stationery *et cetera* the onus thrown on the Honorary Medical Committee, who so promptly dealt with this work,

was extremely great. A very large portion of the accounts they were able to return to the underwriters as "reasonable" without any reference to the doctors concerned. Several reports had to be obtained from doctors mostly on account of inadequate information being supplied, several were reduced and several increased. While it was found that a few medical men were attending longer or charging more than the Committee thought reasonable, on the other hand doctors were charging less fees than they should.

A rearrangement has been made that only definite accounts not according to schedule, are to be forwarded for revision. I think the atmosphere has been very greatly cleared both with the insurance companies and the profession.

The Special Medical Committee consisted of the late Dr. Trethowan, Dr. Juett and Dr. Anderson and now as Dr. Juett has gone to England, Mr. Hadley and myself have been appointed to the vacant places.

While asking you to realize the very great strain that has been placed on this Committee, I am glad to report the very loyal support given to it and its decisions by the bulk of the members of the profession.

Unfortunately all practitioners are not members of the Branch and although the Committee made recommendation regarding some of the accounts of non-members, we could not do more than comment on their charges in some cases and insurance companies were compelled to pay accounts that all of you would think unduly high.

Benevolent Fund.

This much needed Branch of the Association was successfully started this year and I am pleased to report I have had good support from several members. I would like, however, the Association as a body to take the matter up whole-heartedly and every member to give something to those in need in a practical way by sending in a subscription at once. The fund now has £160 in hand and I will ask you tonight to adopt the rules and regulations governing it.

Auditors.

You will be asked to elect two auditors for the ensuing year. The present auditors are Dr. A. E. Randell and Dr. S. E. Craig, for whose valuable services last year I tender the thanks of the Council.

Dr. Crisp's Retirement.

Before closing I would like to say how much the Council regrets Dr. Crisp's decision not to stand again this year for election as Honorary Secretary and on behalf of the Association I desire to tender him sincere thanks for his six years' valuable work.

I would like also to thank the Council and the Association for the loyal support they have given to me during the year as President. I cannot vacate the chair without again thanking you for the honour you have conferred on me of having me for your President during the past year. I fully appreciate the honour.

I feel that I have always worked in the interests of the Branch and point with pride to having founded the Medical Defence Association which has been of greater value than most of you know and which has funds amounting to £320 and the Medical Benevolent Fund which is well established and is going to prosper.

One cannot help commenting on the remarkable improvement in the attainments of the profession of Western Australia during the past ten years. I do not mean that as a criticism of those practising prior to ten years ago, but it is worthy of note that so many of our younger members have explored every avenue to make themselves thoroughly proficient in their profession before asking the public to seek their advice.

Their efforts are worthy of reward and I hope they receive the full appreciation of the profession and the public.

The standard of conduct in Western Australia is high and it is the duty of the younger members to keep it high so that they may have the greater confidence and respect of the public.

In choosing a President of the Branch each year I think the honour should only go to those who have interested themselves in the work of the profession and the Branch and I have much pleasure in introducing to the office of President such a one in Dr. Baldwin Gill.

Election of Office Bearers.

The President then declared the result of the election of the office-bearers and members of the Council for the ensuing year as follows:

President: Dr. H. B. Gill.
Vice-President: Dr. H. J. Gray.
Ex-President: Dr. J. J. Holland.
Honorary Secretary: Dr. L. E. Le Souef.
Members of Council: Dr. T. L. Anderson, Dr. D. P. Clement, Dr. M. K. Moss.

Induction of President.

Dr. J. J. Holland then introduced Dr. H. B. Gill as the President of the Branch.

Librarians' Report.

The Librarians' Report was read by Dr. R. D. McK. Hall and adopted. On the motion of Dr. H. J. Gray it was resolved that the question of the library be referred to the Council for conference with the librarians as to more efficient working.

Constitution of the Federal Council.

The constitution of the Federal Council was submitted with the recommendation by the Council of the Branch. The constitution was agreed to on the motion of Dr. F. A. Hadley, seconded by Dr. J. Bentley.

Election of Auditors.

Dr. A. E. Randell and Dr. S. E. Craig were unanimously elected auditors for the ensuing twelve months.

Post-Graduate Work.

Dr. A. W. Farmer gave notice of his intention to move at the next meeting that a post-graduate week be held.

Ethical Committee.

The question of the constitution was raised and much discussion took place in reference to the insertion of advertisements in the daily press. Dr. Crisp replied that the Ethical Committee was constituted under the regulations in force and that the insertion of advertisements was laid down by the ethical rules.

Medical Benevolent Fund.

Dr. Holland submitted a report on the successful formation of the Medical Benevolent Fund which was adopted.

Psychosis and Gross Brain Lesions.

Dr. R. G. Williams read a paper entitled: "Psychosis Associated with Gross Brain Lesions." This will be published in a subsequent issue.

Vote of Thanks.

The President, Dr. Gill, moved a hearty vote of thanks to Dr. Bentley for his generosity in entertaining the Branch, which was supported by the members present.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Walker, Milner Frederick Elford, M.B., B.S., 1927 (Univ. Sydney), 12A, Karori Flats, Elizabeth Bay.
Spark, Torry Ernest Hester, M.B., B.S., 1926 (Univ. Sydney), Gordon Road, Gordon.
McNamara, Matthew Joseph, M.B., B.S., 1928 (Univ. Melbourne), 30, Park Avenue, Mosman.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Reynolds, Thomas O'Loghlen, M.B., Ch.B., 1930 (Univ. Melbourne), Toowoomba Hospital, Queensland.

Drew, John Frederick Francis, M.B., Ch.B., 1930 (Univ. Melbourne), Launceston Hospital, Tasmania.

Drew, Joseph Harold D'Amer, M.B., Ch.B., 1916 (Univ. Melbourne), 354, Park Street, South Melbourne.

Hill, Nettie Grace, M.B., Ch.B., 1930 (Univ. Melbourne), 95, Princess Street, Kew, E.4.

Davis, Ethel Eva, M.B., Ch.B., 1930 (Univ. Melbourne), Caulfield Convalescent Hospital, Caulfield.

Medical Societies.

THE SYDNEY HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE SYDNEY HOSPITAL CLINICAL SOCIETY was held in the Maitland Lecture Theatre, Sydney Hospital, on March 5, 1930, DR. C. E. CORLETTE, the President, in the chair.

Malignant Disease of the Prostate.

DR. R. H. BRIDGE showed a patient who had suffered from malignant disease of the prostate gland. This patient had been treated by means of radium, five three milligramme needles having been inserted radially into the bladder and one ten milligramme needle in a gold tube having been placed into the prostatic urethra. Under treatment the prostatic condition had entirely cleared up, but a swelling had recently appeared in the region of the right frontal sinus. Dr. Bridge considered that this swelling might be the result of a metastasis.

Dermatological Conditions.

DR. G. R. HAMILTON showed two patients. The first was suffering from multiple lipomata, the tumours of the forearms being very prominent.

The second patient was extensively tattooed. This patient's serum yielded a positive reaction to the Wassermann test.

Multiple Injuries.

DR. H. SKIPTON STACY showed a patient who was suffering from the effects of multiple injuries. A full account of this case will appear in a subsequent issue.

Asthma.

DR. E. H. STOKES showed a patient who was suffering from asthma. This patient had not given reactions to the various proteins to which it was suspected he might be sensitive. His nose and throat had been investigated. No focus of infection had been found in that region. His sputum contained streptococci. Small doses of a vaccine prepared from his sputum had apparently excited asthmatical paroxysms. At the suggestion of various members, it was decided to continue the administration of the vaccine, using very small doses. The suggested initial dose was 0·06 mil (one minim) of a vaccine containing five thousand streptococci per cubic centimetre.

Disseminated Sclerosis.

DR. WILFRED EVANS showed a married man, aged forty-eight years, who had complained of weakness of the right leg and unsteadiness for two years. On examination he was unable to move the eyes above the horizontal plane and there was definite nystagmus. The cranial nerves otherwise were intact, but speech was definitely scanning. His gait was of the spastic ataxic type. Rombergism was present and there was irregular breaking of movement at the end of the finger-nose test. The right leg was spastic and there was considerable weakness of the flexor muscles of the foot. Vibration sense was lost in the lower extremities; all other sensations were normal. Ankle clonus, exaggerated knee jerk and the Babinski phenomenon were present on the right side. The deep reflexes throughout were hyperactive, the abdominal reflexes absent. The pupils reacted to light and accommodation. Bitemporal pallor of the optic discs was present. The Wassermann test did not yield a reaction.

Dr. Evans said that the interesting feature in this case was the presence of a localized patch of sclerosis in the mid-brain causing paresis of both eyes above the horizontal plane.

Bronchiectasis.

Dr. Evans also showed a single woman, twenty-two years of age. Her history was that of chronic cough since an attack of pneumonia at nine years of age. Her tonsils and adenoids had been removed five years later without any appreciable effect on the symptoms. She expectorated about one pint of foul-smelling sputum every day and was losing weight.

Physical examination revealed definite clubbing of the fingers and an area of dulness at the base of the right lung, with diminished breath sounds and fine crepitations.

Examination of the nasal sinuses had revealed nothing abnormal. "Lipiodol" had been injected into the lungs by the crico-thyreoid route on February 27, 1930, and X ray examination following this had revealed a saccular bronchiectasis at the right base. The intention with regard to treatment was to perform a right-sided pneumothorax and phrenicotomy.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the Adelaide University on November 8, 1929.

Liver Treatment in Anæmia.

DR. E. MCLAUGHLIN exhibited a number of charts illustrating the response to liver treatment in cases of pernicious anæmia. The response in no way varied from that described by Minot and Murphy in their original article. He stated that the form of the reticulum varied according to the percentage of reticulocytes present. As the reticulocytes approached their greatest concentration, the loose open reticulum became more condensed, usually taking the form of a rounded or band-like mass. As the percentage again fell, the open reticulum was seen.

Virus Diseases of Plants.

MR. G. SAMUEL gave a brief review of some recent work on methods of inoculation in plant virus diseases, especially that published from the Boyce Thompson Institute for Plant Research in New York State. This work which dealt with tobacco mosaic disease, had shown that the older methods of inoculation of viruses by needle injections, scratches or rubbing with wounding, were not so efficient as an extremely light rubbing of the virus over the leaf without producing any wound other than the breaking of hair cells. The latter method, moreover, allowed the development of "local lesions" at the point of entry of the virus to be seen, whereas the wounding associated with the older methods of inoculation had tended to obscure or distract attention from these. The development of local lesions within a period of some four or five days and the large numbers which could be produced on a single plant by a concentrated virus, allowed much greater economy of time and space and also provided a much more convenient method for the quantitative comparison of the potency of different samples of virus than the old methods whereby one had to wait for systemic infection of the whole plant after an incubation period of some fourteen days and had to work with large numbers of plants for any reasonable degree of accuracy.

Inoculation with a very dilute sample of virus might result in the production of only one local lesion on a leaf. This was found to give rise later to the usual systemic infection just as well as if there had been larger numbers

of primary lesions. Such cases provided an interesting field for speculation as to whether a "pure line" of virus had been isolated, resulting from the entry of a single virus particle at the site of the local lesion.

Mr. Samuel then gave an account of work at the Waite Institute on inoculating methods with the virus disease known as tomato wilt which tended to confirm the results secured at the Boyce Thompson Institute.

Post-Graduate Work.

POST-GRADUATE COURSE IN ADELAIDE.

ATTENTION is again called to the post-graduate refresher course to be held in Adelaide from May 25 to 29, 1930. The full programme was published in the issue of April 12, 1930. The fee for the course is two guineas. Members are reminded of the two lectures to be delivered by Sir Richard Stawell, on Wednesday, May 28, at 8.30 o'clock p.m. on inflammatory conditions of the brain, and on Friday, May 30, on coronary thrombosis. The lectures are free to members of the course, but any member of the British Medical Association may attend on payment of one guinea. Tickets may be had on application to the Honorary Secretaries, Dr. S. R. Burston and Dr. F. N. Le Messurier.

LECTURES IN MELBOURNE.

THE Melbourne Permanent Committee for Post-Graduate Work has arranged with Professor Evarts Graham, Professor of Surgery, Washington University School of Medicine, St. Louis, United States of America, to deliver a course of six lectures in Melbourne during the latter half of July, 1930.

Professor Graham is known in Australia chiefly in connexion with his work on diseases of the gall bladder and on cholecystography. He has also contributed largely to the advancement of surgery of the chest. Full details of the lectures are not yet available, but it is expected that three lectures will be devoted to the biliary tract and three to thoracic conditions.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on May 5, 1930.

The following degree was conferred *in absentia*:

Bachelor of Surgery: Norman Arthur Walker.

The Diploma in Journalism was awarded to Kenneth Hutton Wilkinson.

The following appointments were approved:

Dr. T. Schenk, Dr. T. J. Connolly and Dr. G. Halloran as Honorary Demonstrators in Anatomy.

Mr. A. R. Woodhill as McCaughey Lecturer in Entomology in the Faculty of Science.

Dr. Margaret Harper as Lecturer in Mothercraft in the Department of Obstetrics.

Correspondence.

TONSILLECTOMY.

SIR: In a pamphlet on tonsils, sent to many members of the Western Australian Branch of the British Medical Association, Mr. W. Kent Hughes says some disasters from hæmorrhage in the practice of a brilliant surgeon were due, in Mr. Hughes's opinion, to the use of a crushing cutting guillotine. As I am an advocate for the use of such a guillotine, would Mr. Hughes be good enough to give reasons for his characteristically emphatic and unsupported statement? In my experience a bleeding vessel cut by the crushing cutting guillotine does not differ from one cut by a knife or scissors or one torn by the blunt "reverse" guillotine. With bleeding from the latter, I know Mr. Hughes has had experience.

Yours, etc.,

H. J. GRAY.

195, St. George's Terrace,
Perth.
April 29, 1930.

AN INTRODUCTION TO A PRACTICE.

SIR: I venture to ask your readers to "look upon this picture and on that." I have recently sold my practice in a Sydney suburb. Introduction of my purchaser was effected by letter to whom he might select, and personally to seventeen others. With the help of my patients' address book and a map, Dr. Purchaser at once commenced a round of calls, "to back your letters," upon every household within my professional connexion. Before I had left the practice and before he called upon his new colleagues, he commenced this "canvass," as he himself called it. "I want you to promise to come to me if you or any of your household get ill. I want you to canvass for patients for me among your friends" were usual requests on personal introduction by me. "If you will canvass a few patients for me among your friends," said he to a plumber, "I will give you the job" of installing a new bath-heater. Such is my first picture. The artist is trying to place it in a plain professional oak frame I left behind me.

And now for the other. Mrs. Purchaser is at the back door of her new home; I am about to enter it from the garage. To her comes friend baker; a punctual, decent fellow is baker, who has served me continuously, though I do not yet know his name; he is merely the carter. Says he: "I have served the doctor here for some years; may I serve you?" "I have already arranged with some one or other!" (I could hear the sniff.) "I will inquire about you. I may give you a turn next month" is the reply. Exit baker, whispering to me, "Will she?" (I swear I could hear his sniff.) As I entered the back door Mrs. Purchaser asked me: "Is he a patient? Is he the man who makes the bread?" I was not questioned as to the quality of the bread. Such is the second picture; the frame has been gilded; I cannot see the oak. Who can?

About one hundred letters were sent. When asked, I refused to assist in the "canvass" by calling with the canvasser.

Yours, etc.,

"SENEX PROMISSA BARBA HORRENTE CAPILLO."
(With my hair standing on end.)

May 6, 1930.

ASPHYXIA PALLIDA.

SIR: An article which is published, although possessing many merits *per se*, will frequently enlarge its scope if suitable comment arises.

It is with this attitude of constructive criticism that Dr. Cuscaden's article in THE MEDICAL JOURNAL OF AUSTRALIA of December 21, 1929, is discussed.

The main tenor of the article was the effect of 8% carbon dioxide gas in oxygen upon the condition called *asphyxia pallida*. This condition is really not a true asphyxia, but is a state of shock, the muscles are flaccid, the fœtus is cold and the capillaries are dilated, forming a large blood pool and therefore a very low blood pressure.

In the case of *asphyxia livida* the administratiqon of carbon dioxide and oxygen is almost the main requirement when the air passages are cleared, but in *asphyxia pallida* it would cause some slight stimulation of the vasomotor centre, provided the gases passed through the

alveoli into the blood stream. The resulting slight vaso-constriction of the arterioles would increase the blood pressure slightly, but this is insignificant in comparison with the result of "Pituitrin" injection which constricts the dilated capillaries.

For some time a 5% mixture of carbon dioxide in oxygen has been used for local researches. The effect upon the fœtus with a good pulse has been highly satisfactory, because the respiratory hormone (carbon dioxide) stimulates the respiratory centre and normal respiratory excursions result.

The most satisfactory treatment of *asphyxia pallida* which the author knows, is given hereunder:

1. The child is held up by the feet according to the method of Prochownick and the body wrapped in warm moist towels; with the infant in this position the respiratory passages are cleared with a catheter.
2. "Pituitrin" is injected deeply into muscular tissue, the amount being 0·3 cubic centimetre of the surgical "Pituitrin."
3. The infant is given twenty cubic centimetres of whole blood derived from the mother or father. It is unnecessary to group the parental and infant bloods as either paternal or maternal blood is satisfactory. The infant is prepared and a wide bore needle is introduced in the subscapular region. This done, another needle is inserted into the donor's vein and the required blood withdrawn. The needle is then withdrawn and discarded and the syringe is attached to the needle previously introduced, in the subscapular region, and the blood is then run into the subcutaneous tissue; the needle is withdrawn and the puncture wound sealed.
4. After the air passages have been cleared, a catheter is passed, if possible, into the trachea. This is preferable to a nasal catheter which allows the air to enter the stomach. Using a gas mixture of 5% carbon dioxide in oxygen, the lungs are inflated at a low pressure every three seconds. This is done by releasing a clip attached to the tubing connected with the reservoir. At the same time the expiratory movement of the Sylvester method is made. The clip is then shut and the expiratory movement is made to empty the lungs.

These measures have met with satisfactory results, but owing to their fortunate infrequency no report of a large series of cases can be offered.

The methods here suggested are merely the result of the application of physiological processes to pathological conditions.

The laboratory research work was done in the Department of Human Physiology and Pharmacology at the University of Adelaide.

Yours, etc.,

R. FRANCIS MATTERS, M.D., F.R.C.S. (Edin.),
Lecturer in Human Physiology and Pharmacology and Tutor in Obstetrics, University of Adelaide.

Adelaide.
May 7, 1930.

DIATHERMY OF TONSILS.

SIR: Though not an authority on tonsils, I have treated a few cases with diathermy and if my own or any in my family had to be removed, no amount of argument would persuade me to have them operated while a diathermy machine was available.

Yours, etc.,

L. CRIVELLI.

32, Collins Street, Melbourne.
May 12, 1930.

Obituary.

DAVID TYRRELL KEYES.

DR. DAVID TYRRELL KEYES who died on March 8, 1930, at Leura, New South Wales, was thirty-seven years of age. He was educated at Saint Patrick's College, Ballarat. He graduated in medicine at the University of Melbourne in 1916. He served in France, but was invalided home in 1917 after suffering from an attack of pneumonia. He practised in Shepparton for a short time, but on his father's death took over his practice in Nathalia. This he afterwards sold and went to Tibbooburra. He developed pulmonary tuberculosis early in 1926 and was ordered to the Blue Mountains in October of that year, where he recovered sufficiently to enable him to start practice in Tullamore in January, 1928. He broke down during the following year and was in hospital until the time of his death. He was the second son of the late Dr. F. J. Keyes, of Victoria, and a brother of Miss Letty Keyes, a well known pianist.

CYRIL SHEPHERD.

WE regret to announce the death of Dr. Cyril Shepherd which occurred at Sydney on May 14, 1930.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned has been registered under the provisions of *The Medical Act*, 1918, of Tasmania, as a duly qualified medical practitioner:

Duncombe, Cedric, M.B., B.S., 1922 (Univ. Melbourne), F.R.C.S.E., 1929, Hobart.

VICTORIA.

THE undermentioned have been registered under the provisions of Part I of *The Medical Act*, 1928, of Victoria, as duly qualified medical practitioners:

Murray, Alan Ambrose, M.B., B.S., 1930 (Univ. Melbourne), c.o. Dr. Kemp, Mont Albert Road, Mont Albert, E.10, Melbourne.
Brydon, Adam Gibson, M.B., Ch.B., 1900 (Univ. Melbourne), M.D., 1918 (Univ. Edinburgh), 12, Collins Street, Melbourne.
Claridge, Henry Arthur Herbert, M.B., B.S., 1892 (Univ. Durham), 3, Blair Street, Coburg, N.13.
Glastonbury, Kevin, M.B., B.S., 1926 (Univ. Adelaide), Victoria Street, Ballarat.
Vine, James Miller, M.B., B.S., 1924 (Univ. Melbourne), c.o. James Miller and Company, 357, Little Collins Street, Melbourne, C.1.
Baker, Aubrey Wilfred, M.B., B.S., 1930 (Univ. Melbourne), 18, Court Street, Box Hill, E.11.
Davis, Ethel Eva, M.B., B.S., 1930 (Univ. Melbourne), 313, Elizabeth Street, Hobart.
Drew, John Frederick Francis, M.B., B.S., 1930 (Univ. Melbourne), 7, Palmer Street, Jolimont, C.2.
Hill, Nettie Grace, M.B., B.S., 1930 (Univ. Melbourne), 95, Princess Street, Kew, E.4.
Paterson, James Hudson, M.B., B.S., 1930 (Univ. Melbourne), 40, Marine Parade, St. Kilda, S.2.
Philpott, Jack Melville Curran, M.B., B.S., 1930 (Univ. Melbourne), 214, Part Street, West Brunswick, N.12.
Reynolds, Thomas O'Loghlen, M.B., B.S., 1930 (Univ. Melbourne), 15, Trafalgar Road, Camberwell, E.6.
Vincent, Frank Robertson, M.B., B.S., 1930 (Univ. Melbourne), Western Beach, Geelong.
Webster, Victor Henry, M.B., B.S., 1930 (Univ. Melbourne), 10, Newlyn Street, Caulfield, S.E.8.

Additional registration:

Colquhoun, John Boyd, F.R.C.S., 1929 (Edinburgh).
Buchanan, Andrew Russell, M.D., 1930 (Melbourne).
Lockwood, Lionel, M.D., 1930 (Melbourne).
Cantor, Cecil Nathaniel Love, M.D., 1930 (Melbourne).

Frank, Theophil Johannes Friedrich, M.D., 1929 (Melbourne).
Ellery, Reginald Spencer, M.D., 1929 (Melbourne).
Phillips, Henry Anthony, M.D., 1929 (Melbourne).
Worcester, Reginald George, M.D., 1929 (Melbourne).

THE ROSS AWARD FUND.

WE have received from an anonymous donor in New Guinea the sum of five pounds for the Ross Award Fund. This fund was started last year in order to give some recognition to Sir Ronald Ross for his discoveries on the mode by which malaria is carried and for his efforts in the prevention of the disease. The money has been sent to London.

Books Received.

OUR NEW RELIGION: AN EXAMINATION OF CHRISTIAN SCIENCE, by the Rt. Hon. H. A. L. Fisher, F.R.S.; 1929. London: Ernest Benn Limited; Sydney: Angus and Robertson. Demy 8vo., pp. 191. Price: 7s. 6d.

ANNALS OF ROENTGENOLOGY: A SERIES OF MONOGRAPHIC ATLASES, edited by James T. Case, M.D.; Volume X, "Peptic Ulcer," by Jacob Buckstein, M.D.; 1930. New York: Paul B. Hoeber, Inc. Demy 4to., pp. 360, with two hundred and forty Roentgen ray studies and forty-seven clinical illustrations. Price: $12.00.

THE DRAMATIC IN SURGERY, by Gordon Gordon-Taylor, O.B.E., M.A., F.R.C.S.; 1930. Bristol: John Wright and Sons Limited; London: Simpkin Marshall Limited. Royal 8vo., pp. 88, with 40 illustrations in the text, 11 of which are fully coloured. Price: 12s. 6d. net.

Medical Appointments.

Dr. E. G. Schwartz (B.M.A.) has been appointed Government Medical Officer at Walgett, New South Wales.

* * *

Dr. F. Trenerry has been appointed to act as Medical Inspector of Seamen pursuant to the provisions of Section 123 of the *Navigation Act*, 1912-1926.

* * *

Dr. J. W. Rollison (B.M.A.) has been appointed Resident Medical Officer, Northfield Mental Hospital, also Visiting Medical Officer, Yatala Labour Prison, Division 2, South Australia.

* * *

Dr. I. G. Hooper (B.M.A.) has been appointed Government Medical Officer at Killarney, Queensland.

Diary for the Month.

MAY 23.—Queensland Branch, B.M.A.: Council.
MAY 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 28.—Victorian Branch, B.M.A.: Council.
MAY 29.—New South Wales Branch, B.M.A.: Branch.
MAY 29.—South Australian Branch, B.M.A.: Branch.
JUNE 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
JUNE 3.—New South Wales Branch, B.M.A.: Post-Graduate Work Committee.
JUNE 4.—Victorian Branch, B.M.A.: Branch.
JUNE 5.—South Australian Branch, B.M.A.: Council.
JUNE 10.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 12.—Victorian Branch, B.M.A.: Council.
JUNE 12.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 13.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xvi.

THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 21, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company, Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
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